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ON THE INSENSIBLE PERSPIRATION AND ITS CLINICAL SIGNIFICANCE.

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INTRODUCTORY AND HISTORICAL.

IN working on problems of metabolism we are accustomed to make our calculations starting from the side of the intake (that is, oxygen consumption) from which we calculate the heat production expressed in calories. These figures must under basal conditions approximately equal the heat loss, except for the small amount of energy which is converted into work (heart action, respiration, intestinal activity). Until recently it has been difficult, if not almost impossible, in clinical research to do any work on this side of the equation—namely, the heat loss—which is made up chiefly of three factors: radiation, convection and vaporization. We have fairly reliable information regarding the proportion which these three factors bear to one another in the total heat loss—namely, for radiation about 60% to 65%, for convection about 10% to 15%, and for vaporization about 25%; but the relation of these figures changes with alterations in the environment (temperature, humidity). That it depends much on the surroundings is clear from such a consideration as, for example, the following—that a cold wall will absorb more heat from a human body than a wall of the same temperature as the body. On the other hand, the body reacts to differing external temperatures by either contraction or dilatation of the cutaneous vessels, and consequently different amounts of heat are supplied to the skin, with resulting changes in skin temperature. If the experimental conditions can be kept constant so that the proportion of the three factors remains constant too, there is a possibility of finding out the amount of radiation, as both convection and the rate of vaporization can be measured. The sum of these three factors must be equivalent to the total number of calories produced as measured by one of the usual methods. This is one of the reasons why work on vaporization is interesting at the present day and promises valuable results apart altogether from clinical research.

Vaporization.

The following discussion deals only with the factor of vaporization. The vaporization of water from the body takes place by means of the insensible perspiration. This has clearly to be distinguished from sweating. It means the discharge of water in the form of vapour only, so that it is insensible and invisible, but weighable. The term insensible perspiration is used by most authors to mean the discharge of water from the skin only; but as all work on insensible perspiration is done by weighing the body, the conditions are much more complex, because by the use of the weighing method the loss of water through the respiratory passages will be determined at the same time, and also the difference in weight between the carbon dioxide exhaled and the oxygen absorbed. The magnitude of the last-mentioned factor depends upon the respiratory quotient and is normally under basal conditions about three grammes per hour. The loss of water from the respiratory passages has often (Osborne[19][20]) been reckoned from the respiratory volume and the amount of vapour contained in the inspired and expired air. As it can be assumed that the expired air is always saturated with moisture, the amount of water discharged by respiration can be determined directly when the respiratory volume and the water content of the inspired air are

known. The figures for water loss by the respiratory passages found in this way are between eight and fourteen grammes per hour. When these two sets of figures (three grammes and eight to fourteen grammes) are known and subtracted from the total weight lost, the rest of the total loss of weight is to be attributed to the water loss from the skin, and provided that sweating has been excluded, this figure represents the value for the insensible perspiration *in sensu strictiori*. But on account of the technical difficulties many authors define insensible perspiration, not as this part of the loss in weight, but as the total loss in weight, which definition will be maintained in the following discussion.

Insensible Perspiration.

Many workers have contributed in the last fifteen years to the subject of insensible perspiration. The first and most important modern work is that of Benedict and his co-workers, Benedict, Root and Wardlaw.[1][2][3] The figures which they obtained are still the most important ones we have, and they are confirmed by most of the later workers, such as Jores[9][10] and Whitehouse, Hancock and Haldane.[30]

On the average, Benedict, Benedict, Root and Wardlaw found an hourly loss of 25 to 35 grammes—that is to say, about 700 grammes *per diem*. Out of the total loss of weight, about 65% is to be attributed to loss from the skin and about 35% to loss by the lungs. But these figures are not constant, as the water lost by the lungs depends to some extent upon the humidity of the inspired air. On the other hand, recent experiments by Japanese workers (Kuno[11]) show that there is a fixed relation between water loss from the respiratory passages and that from the skin, an increased loss by the lungs being accompanied by a diminished loss from the skin, so that the total loss by both organs remains fixed, even if the proportion "loss through lungs to loss through skin" varies from 1:3·3 to 1:2 according to whether very wet or extremely dry air is inspired.

The evaporation at 37° C. of one gramme of water requires an energy consumption equivalent to 0·58 Calorie. This loss of heat from the surface of the body has to be made good by a corresponding increase in heat production. A daily loss of about 600 to 700 grammes by insensible perspiration would therefore mean a loss of 350 to 400 Calories a day.

Mechanism of Insensible Perspiration.

In order to appreciate more clearly what these figures mean and the possibility of controlling the factors concerned, it is necessary to discuss briefly the mechanism of insensible perspiration, which until recently has been imperfectly understood. In the past there has been much controversy over the mechanism of the water loss from the skin. Schwenkenbecher[29] ascribed insensible perspiration almost entirely to a form of invisible secretion of the sweat glands. On the other hand, Loewy and Wechselmann[12] and their co-workers held that the whole cutaneous insensible perspiration was produced without the work of the sweat glands, as they were able to show that for a subject suffering from congenital absence of sweat glands approximately the same figures for insensible perspiration were obtained as for normal persons. Whitehouse, Hancock and Haldane[30] supported this view; but most recent investigators hold intermediate views. Recently this question has been solved by the experiments of Kuno,[11] who showed that the cutaneous insensible perspiration is due (i) to a physical process—probably the diffusion of the tissue fluid through the epidermis—and (ii) to a certain degree to the activity of certain sweat glands which are numerous in the region of the palms and the soles and which are continuously secreting invisible sweat. It must therefore be concluded that the cutaneous insensible perspiration is due, not only to the discharge of water through the epidermis (osmosis), but also to the perpetual invisible—that is, "insensible"—secretion of a little sweat in certain small areas of the body.

In view of these findings it is now understandable that the insensible perspiration cannot always be constant, as the activity of glands will be much affected by the external temperature and by certain nervous influences, while the osmotic discharge of water through the epidermis can be influenced, not only by the temperature and humidity of the surroundings, but by the temperature of the skin itself. Benedict[3] does not attribute an important influence to the surrounding temperature, provided that the surroundings are "comfortable". He and some other workers maintain that the insensible perspiration is independent of the subject's being dressed or naked. On the other hand, Kuno[11] noticed, in special experiments in which the influence of a gradual rise of room temperature was studied, that the cutaneous insensible perspiration increased by as much as 100% in response to a rise of room temperature. Lately Moog[15][16][17] found that the compression of the veins of a limb diminished the insensible perspiration and that hyperæmia caused by yohimbine or amyl nitrite increased the rate of insensible perspiration while adrenaline decreased the rate. This means that the insensible perspiration is dependent on the blood circulation through the skin, which is regulated reflexly through the nerves conveying thermal sensibility acting upon the heat-regulating centre. (The mechanism of the direct action of the temperature of the blood upon the heat-regulating centres is not entered into in the present discussion.) The reaction of the smaller blood vessels is the most important factor in the determination of heat loss and is therefore the factor which varies most with changes in metabolism.

Previous investigations by Rubner[28] showed that the insensible perspiration greatly increased (by about 50%) after meals—a fact later confirmed by Benedict and Root[3] (11% to 27%). Jores[10] found an increase in the insensible perspiration of 50% to 60% after walking, and Meyer found a more considerable increase as the result of the restlessness of infants. We have now seen that temperature as well as food and exercise affect the insensible perspiration, factors which are the same as those known to influence the basal metabolism.

Insensible Perspiration and Metabolism.

Benedict[1][2] tried to prove that the rate of insensible perspiration (total weight loss) was always proportional to the basal metabolic rate. By testing the basal metabolism and the insensible perspiration at the same time, he found that the values obtained always bore a constant and linear relation to one another, so that he could construct a graph from which the values for the hourly rate of insensible perspiration could be read off when the rate of heat production per twenty-four hours was known. He also showed that the insensible perspiration of different subjects, though varying greatly, was proportional to the body surface as calculated from the height and weight (Du Bois table). Many later investigators confirmed this important statement, most of them obtaining figures which were at least within 10% to 20% of the former Benedict figures. This very close relation between insensible perspiration and basal metabolic rate seems to be possible only if the proportion of the loss of heat by vaporization of water (that is, insensible perspiration) to the total energy consumption as determined from the basal metabolic rate is fairly constant. Recently Soederström and Du Bois found that the heat eliminated by evaporation covered about 24% of the total heat loss of normal subjects under "comfortable" temperature conditions, and that during a rise in temperature the rate of vaporization increased and the rate of radiation decreased. This fact once more confirms the importance of the role of the temperature in experiments on insensible perspiration.

Experiments on Insensible Perspiration.

An attempt was made to determine whether the method was practicable for experimental and clinical work and what special sources of error were important and to be avoided also how the results obtained under the rather

hot climatic conditions prevailing in Sydney compared with those obtained elsewhere, since nearly all previous papers report the results obtained in temperate climates.

Technique.

In the present investigation the Sauter balance was employed; it was specially made for such experiments and has been used by nearly all workers in this field during the last fifteen years. The balance has an accuracy of 1 in 1,000,000 (less than 0·1 gramme in 100 kilograms). The technique followed was that described in the papers of Benedict, Wardlaw and Jores. As the modern apparatus is made entirely of iron, there is no longer any danger that hygroscopic material will interfere with the experiments by absorbing water. All sheets and clothing used during the experiments were always kept in the special weighing room, which was maintained at a constant temperature and approximately at the same humidity. Temperature and humidity were controlled by means of dry bulb and wet bulb thermometers. The patients undressed before entering the weighing room and put on pyjamas (sometimes also socks). It is very important to prevent patients from taking a bath or a shower within some hours before the experiment; wet skin or wet hair upsets all experiments, because the evaporation of this extraneous moisture cannot be controlled.

Following the advice of Benedict, a rubber mattress covered by a sheet was used, the patient being always dressed in pyjamas and covered by a second sheet. But during the first month of experiments it was found that this technique was not reliable, as it was discovered that a rubber mattress prevented the normal evaporation from the skin, and therefore the figures obtained in these experiments were too low and could not be considered normal figures for the insensible perspiration. To illustrate this the figures from two experiments may be quoted:

Experiment I.—Without a rubber mattress the hourly loss by insensible perspiration was 29·1 grammes. With a rubber mattress the hourly loss by insensible perspiration was 22·2 grammes—24% below normal.

Experiment II.—Without a rubber mattress the hourly loss by insensible perspiration was 27·8 grammes. With a rubber mattress the hourly loss by insensible perspiration was 20·5 grammes—25% below normal.

It may be that the low temperature of the mattress influences the result; but even after the rubber mattresses were warmed, these figures remained unchanged. Therefore, in subsequent experiments mattresses or sheets made of india-rubber were discarded.

Good ventilation is necessary, but all draught has to be prevented, as it always affects the results; again, the very sensitive scale will not work reliably in a draughty room. The relative humidity did not appear to make any appreciable difference to the results so long as it was not under 25% or over 65%, and provided that the temperature was kept at about 23° C. In the experiments here recorded the humidity was maintained at about 50%.

As only the insensible perspiration was under investigation, it was extremely important to make sure that no sweating occurred during the experiment and therefore the back and axillæ, hands and soles of the patient had to be carefully watched during and after the experiment. In a few cases, the subjects being chiefly of the nervous type, the "sensible" sweating of the palms and soles could not be stopped. In these cases rubber gloves were used and the soles were covered with pieces of rubber inserted into the stockings; the evaporation of sweat was thus prevented. The amount of water which was prevented from evaporating in this way was between 12·5% and 17% of the total amount of the insensible perspiration. Figures obtained in this way were reliable, as they were always constant.

All figures recorded are based on observations upon normal subjects under "basal" conditions; the subject has his last protein-free meal early the previous evening; he is brought on a stretcher to the weighing machine, or if he has come on foot, he lies down for from forty to sixty minutes. As different persons react differently, no definite waiting time can be given for the establishment of stable conditions. The easiest way to find whether stable conditions have been attained is to start the experiment after half an hour's rest on the weighing machine and to wait until the figures for the times taken for each five grammes of lost weight become constant in three successive weighings.[1] This sometimes takes up to an hour. The figures having become constant, the experiment is continued for another thirty to sixty minutes. The patient must remain in a state of complete rest; there must be practically no movement, and no talking is allowed.

A complication is, however, introduced by the fact that if the patient falls asleep lower values for insensible perspiration are obtained within about ten minutes after the onset of sleep. Table I shows that restlessness may readily increase the insensible perspiration by over 10%, and that a short period of sleep decreases it by about 8% to 25%. All later experiments showed that the day sleep causes a decrease of about 15% in the normal figures for insensible perspiration—an effect which is not in accordance with the statement of Benedict and Wardlaw[(2)] that day-time sleep does not interfere in any way with the normal experiments and that only the night sleep influences (by about 15%) the rate of the insensible perspiration. To obtain constant figures I therefore always prevented the patient from falling asleep.

Temperature Factor: Cooling Experiments.

The most important question to be settled in all investigations of this kind is whether the surrounding temperature influences the insensible perspiration in any way. As mentioned above, most previous workers,

[1] The following method of observation was used. The subject having been placed in the recumbent position on the "bed" of the Sauter balance, the machine is approximately put into balance by means of heavy weights, the more accurate adjustment being made by lead shot, so that the initial weight on the side of the subject is about two or three grammes greater than on the opposite side. The scale swings so slowly that a full movement takes about a minute. The movement of the pointer is recorded and as soon as it moves equally to both sides the exact time is recorded (or a stop-watch is started). A weight of five grammes is now put on the weighing tray over the bed—that is to say, on the side of the patient. As soon as the pointer's movements are once more equal on both sides the time is recorded. This time indicates the time required for the loss of five grammes of body weight.

TABLE I.
Effect of Sleep.

Subject.	Age. (Years.)	Square Metres of Body Surface.	Calories per Day.	Insensible Perspiration. (Grammes per Hour.)			Remarks.
				Awake.	Asleep.	Decrease. (Percentage.)	
C	14	1·53	1,400	33·0 29·1	26·0–23·5	10–19	Restless. Quieter.
H	19	1·72	1,624	39·3	31·8	20	
G	23	1·78	1,700	40·6	30·7	25	
A	25	1·90	1,775	38·1	35·3	8	

especially Benedict,[3] did not attribute much importance either to the skin temperature or to the surrounding temperature. They stated only that a "comfortable" feeling was indispensable. This is rather vague, as nearly everybody can become accustomed to lower temperatures without feeling cold or uncomfortable, and yet these conditions may be quite sufficient to influence the insensible perspiration. Indeed, some recent investigators (Whitehouse[28]) hold that cooling of the skin has a pronounced effect upon the rate of insensible perspiration. The most recent work is that of Kuno,[11] who studied the changes in insensible perspiration in small areas of the skin and not on the whole body, so that such experiments are not comparable with those of other workers. In view of the apparent discrepancies between the findings of previous workers, it was deemed desirable to carry out a series of experiments, in order to observe whether cooling had any pronounced effect upon the insensible perspiration.

In order to determine the degree of cooling and to obtain reproducible conditions the temperature of the subject has to be recorded. The mouth temperature is of no significance, as it is nearly always constant even when the skin temperature decreases. It may even increase slightly under these conditions. On the other hand, it is difficult to compare the skin temperature in the presence of different surrounding temperatures, as different areas of the skin react quite differently and some parts of the body maintain a fairly constant temperature.

The reaction of the skin to changes in the temperature of the surroundings depends entirely on the response of the small blood vessels of the skin, which, by means of contraction or dilatation, diminish or increase the blood flow through the skin and regulate by these means the temperature of the outer layers of the skin. Recent observations of Sheard and Maddock[19][20] prove that the extremities play the greatest part in the elimination of heat and that the hands and feet react much more to heating and cooling than the trunk. The feet are usually considerably colder than the other parts of the body, as their vessels tend to remain contracted, and it is not until the surrounding temperature exceeds 27° C. that vasodilatation begins, and then the temperature of the toes becomes rapidly raised. The hands react in nearly the same way. All other areas react more slowly (the trunk very slowly), although their initial temperature is higher than that of the hands and feet. The forehead has usually the highest temperature of all skin regions and does not cool greatly even in cool surroundings (see Table II). It was frequently observed that the temperature increased here when in all other regions the temperature was decreasing. It is therefore fallacious to rely on the forehead temperature for testing the constancy of the skin temperature, as did Lange[13] and Jores.[9] It is always necessary to take the temperature of different areas of the body surface and then the most suitable method appears to be to calculate an average surface temperature. The suggestions of Pfleiderer,[21] Winslow and Maddock were followed—namely, to take the temperature of eight different fixed points on the body surface and to multiply the figures so obtained by the percentage area of each of the regions investigated in relation to the total surface of the body, adding together all figures and finally dividing the result by 100, thus obtaining the average skin temperature. The temperatures of the forehead, abdomen, chest, arm, hand, thigh, leg and foot were taken by means of a swiftly working dermatherm (thermocouple). Instead of a thermocouple, a small flat mercury thermometer fixed for a short time with pieces of felt to the skin may be used; this is a method (Ipsen) which gives quite reliable results, but is cumbersome.

For control purposes it is necessary to obtain what may be described as normal external conditions. It was found that the best surrounding temperatures to employ in order to obtain constant skin temperatures ranged from 22° to 24° C., the subject being covered with a sheet and wearing light clothing (pyjamas). In rooms with a temperature higher than 26° C. the skin temperature of the extremities rose rapidly and became nearly equal to that of the trunk (see Table II). Under these conditions there is always the danger that sweating may occur, and as we found these conditions to prevail in Sydney between the middle of December and March, experiments during these periods had to be abandoned.

In beginning an experiment it is necessary to realize that a considerable time is required to obtain constant skin temperatures, especially when the surrounding temperature is low, and even at 24° C. the hands and feet remain considerably cooler than the forehead. The usual "average" skin temperature under "normal" conditions is found to be about 33° C., the temperatures recorded in the individual areas being approximately 33° C. for the forehead, 33° or 34° C. for the chest and abdomen, 32·5° C. for the palms, 30° to 32° C. for the legs, 29° or 30° C. for the instep and 28° C. for the soles. It may be of interest to state that as a rule the temperature of the extremities is about two degrees higher in summer than in winter, even when the room temperature is kept the same; this proves that the level of heat regulation is different in summer and in winter.

In Table III some of the results of cooling experiments on 23 normal students can be seen. The slight cooling of a subject by the removal of the covering sheet or pyjamas, or both, in a room with "comfortable" temperature (about 23° C.) causes the average skin temperature to decrease on an average by 2° or 3° C. The effect of this cooling is to decrease the rate of insensible perspiration by about 15% to 20%. This proves that the temperature factor is of the greatest importance and must be carefully controlled

TABLE II.

Skin Temperatures in Degrees Centigrade.

Area.	Mr. H.		Mr. C.		Miss B.		Mr. L.		Mr. E.	
Forehead ..	32·1[1]	34·2	31·6	33·6	33·0	32·1	33·0	33·6	33·2	33·2
Chest	35·0	33·0	33·6	31·6	34·2	31·6	34·2	30·4	34·7	30·7
Abdomen ..	34·5	32·1	34·2	31·6	34·2	30·4	35·0	30·8	35·0	31·5
Hand	32·8	31·6	32·5	30·8	33·6	29·0	32·9	29·1	34·3	31·1
Thigh	33·6	30·8	33·0	28·6	34·2	27·8	34·2	27·8	34·0	30·3
Leg	33·0	30·8	33·0	27·8	33·0	26·3	33·6	26·3	33·2	30·0
Foot	33·6	30·0	31·9	27·0	30·0	25·8	31·6	23·7	33·0	29·5
Sole	33·6	29·2	29·4	26·3	29·4	25·0	30·0	23·7	32·0	28·0
Average skin temperature	33·8	32·0	31·1	30·4	33·3	29·3	33·9	29·3	33·2	30·9
Decrease of insensible perspiration ..	5%		10%		25%		20%		30%	

[1] The first temperature was taken before the experiment, the second after one hour's rest without cover. The room temperature was about 23° C.

in all experiments on insensible perspiration. Therefore the results of all previous work in which the skin temperature was not controlled cannot be regarded as reliable.

As most workers in this field, including Benedict, do not mention this definite effect of cooling upon the insensible perspiration, it may be that young Australians react more noticeably and more rapidly to the changes of temperature than people living in cooler climates. The fact that they are more accustomed to open-air life and spend much time sunbaking on the beaches may help to explain this, perhaps owing to the effect of such habits upon the training of the skin vessels.

In the present investigation the basal metabolic rate was determined immediately before and after cooling of the subject. Only the oxygen consumption was measured (Krogh, sometimes Benedict apparatus). The figures show that slight cooling of the body surface increases the basal metabolic rate by about 3% to 5%. As no shivering or any obvious muscular activity occurred, our results are in agreement with the statement of Gessler,[3][4] Campbell and Hill[4][5]—namely, that the first effect of cooling is to bring into play the purely chemical heat regulation.

As up to the present very little attention has been paid to the foregoing facts regarding oxygen consumption and insensible perspiration following cooling, it may be stated once more (see Table III) that slight cooling of the body of a normal young reactive subject brings into play simultaneously the chemical heat-regulating mechanism (oxygen consumption +5%) and the physical heat-regulating mechanism, of which only one part—namely, the insensible perspiration (−15%)—was under special investigation in the present series of experiments. It may be of interest to mention that when a person has been accustomed to the cooling experiments, the chemical response becomes less pronounced, while the response of the insensible perspiration remains unchanged.

The importance of the temperature factor in experiments upon insensible perspiration having been demonstrated, all experiments performed in winter were carried out in a special room where the temperature was automatically maintained between 23° and 24° C.

The Normal Figures for Insensible Perspiration.

The following results are based upon about 125 experiments performed on 65 normal students, most of them aged between twenty and twenty-three years, the majority of whom had a surface area of over 1·7 square metres. Provided that the temperature conditions were normal, very good agreement as regards the values for insensible perspiration was usually found amongst subjects having the same body surface. As has already been mentioned, it was Benedict who first advanced the opinion that there was a direct relationship between basal metabolic rate and insensible perspiration (measured as total weight loss per hour without any correction), and he published the curve reproduced in Figure I which makes it possible to predict the basal metabolic rate of a subject if his hourly insensible perspiration is known, and *vice versa*. This work has been confirmed by most subsequent workers.

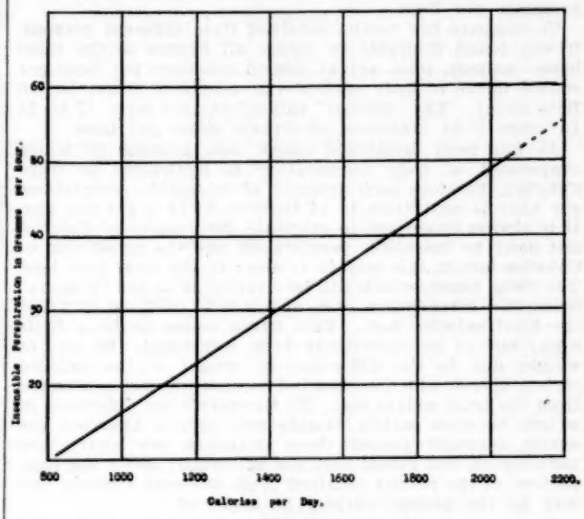

FIGURE I.

Correlation between insensible perspiration (in grammes per hour) and heat production (calories in 24 hours); from Benedict and Benedict.[1]

In more than 80% of the cases here recorded the figures for insensible perspiration did not differ from or were within a range of ±10% of Benedict and Benedict's predicted values; usually the figures for insensible perspiration were about 10% lower than expected. The remaining 20% differed by about 20% from the predicted value. Most of the latter were obtained from tall and heavy persons, whose basal metabolic rate was higher than 1,800 calories per twenty-four hours, and whose insensible perspiration according to Benedict should have been in the region of or higher than 40 grammes per hour. The values actually obtained were, however, invariably lower. The figures obtained from very fat persons were not

TABLE III.

Effect of Cooling.

Subject.	Age. (Years.)	Body Surface. (Square Metres.)	Room.		"Average" Skin Temperature. (Degrees Centigrade.)		Insensible Perspiration. (Grammes per Hour.)			Calories per 24 Hours.			Remarks.
			Temperature. (Degrees Centigrade.)	Humidity. (Percentage.)	Before Cooling.	After Cooling.	Before Cooling.	After Cooling.	Difference (Percentage.)	Before Cooling.	After Cooling.	Difference. (Percentage.)	
L.	20	1·72	24·4	53	33·8	29·5	35·1	26·3	−25	1,707	1,790	+5	
B.	23	1·66	24·0	56	33·3	29·3	31·1	23·1	−25	1,463	1,547	+5·5	
D.	20	1·86	19·5	60	31·3	29·8	38·0	27·5	−27		1,681		
A.	20	1·78	24·5	55	32·7	29·6	29·5	24·3	−19	1,725	1,778	+3	*Diabetes mellitus.*
C.	24	1·71	25·0	50	33·0	30·3	27·8	25·4	−8·5	1,450	1,467	+1	
H.	19	1·72	25·0	50	34·4	32·0	39·3	37·1	−5	1,624	1,732	+6·5	
W.	24	1·85	19·0	70		(i) 29·2 (ii) 29·0	37·5	31·6 (i) 25·0 (ii)	−16 −20 } 33				(i) Cooling of room. (ii) Naked in cool room.
M.	22	1·84	21·5	63		30·1	32·2	28·1[1]	−14				

[1] Experiment performed in reverse manner: (i) cooled, (ii) dressed.

reliable; but it is recognized that the values for the basal metabolic rate of fat persons are also unreliable, as it is impossible to compare heavy persons with good muscles with those whose tissues consist mostly of fat, which is relatively inert, and whose body surface is nevertheless relatively large. For this reason no reliance was placed upon figures obtained from persons with a higher metabolic rate than 1,800 calories per twenty-four hours or on insensible perspiration figures higher than about 40 grammes per hour.

To compare the results obtained from different persons, it was found desirable to reduce all figures to the same basis—namely, total weight loss in grammes per hour per square metre of body surface (as calculated from the Du Bois table). The "normal" values obtained were 17 to 21 (average 19·5) grammes per square metre per hour.

As has been mentioned above, one gramme of water evaporated at body temperature is equivalent to 0·580 Calorie; therefore each gramme of insensible perspiration per hour is equivalent to 14 Calories (0·58 × 24) per day. It is always important to calculate the number of Calories lost daily by insensible perspiration and the proportion of Calories lost in this way in relation to the total heat loss. The value found, which will be referred to as the "relative" insensible perspiration, was about 30% (25% to 32%) of the total calories lost. This figure seems to be a little high; but as has previously been mentioned, the loss of weight due to the difference in weight of the exhaled carbon dioxide and the absorbed oxygen was not subtracted from the total weight lost. As the values for difference in weight between carbon dioxide and oxygen absorbed are nearly constant (about three grammes per hour), the influence of this factor does not materially affect the comparison of the results obtained from different subjects and may for the present purpose be neglected.

Some experiments made on persons after they had taken food showed that the subsequent rise in oxygen consumption was relatively greater than the increase in insensible perspiration.

Effect of Pyramidon and Ultra-Violet Erythema on the Insensible Perspiration.

In order to investigate the manner in which the heat regulator is influenced, use was made of the observation of Gessler,[5][6] that while a pronounced cooling of the body increased the oxygen consumption by about 15% to 18%, this could be prevented by the administration of pyramidon. This drug has no effect on the basal metabolic rate under normal conditions. As the blood temperature remained unchanged, it seemed probable that the effect was brought about through an action upon the nervous system.

The experiments performed confirm Gessler's observation as regards the effect of pyramidon (one gramme in divided doses some hours before the experiment) on the basal metabolic rate. That is to say, the oxygen consumption, which in control experiments without pyramidon was slightly raised by cooling, remained normal or was even slightly decreased after the administration of pyramidon (see Table IV). On the other hand, there was not the slightest effect on the insensible perspiration; the cooling effect remained the same either with or without previous administration of pyramidon. This shows that pyramidon acts only on the chemical heat-regulating mechanism and not on the physical mechanism.

In order to explain the effect of pyramidon—namely, the prevention of the increase in the basal metabolic rate after cooling—it may be supposed that pyramidon acts by peripheral vasodilatation, warming up the endings of the nerves conveying thermal sensibility in the skin so that they would be less influenced by changes in external temperatures. In order to test this hypothesis peripheral vasodilatation was induced by the production of noticeable cutaneous erythema by means of irradiation with ultra-violet rays. The result of the cooling experiment showed that the response to cooling was the same in irradiated as in unirradiated subjects—namely, an increase in oxygen consumption and a diminution in insensible perspiration. While it is possible that a local inflammatory reaction associated with the erythema may interfere with osmotic processes in the skin, this experiment shows that the effect of pyramidon cannot be attributed to the influence of vasodilatation upon the nerves of thermal sensibility. It is therefore probable that the pyramidon acts directly on the heat-regulating centres.

Pathological Cases.

The above-mentioned facts show that the parallelism between the oxygen consumption and insensible perspiration obtains only so long as "normal" conditions prevail and that the range of these "normal" conditions is limited. From this point of view, it seemed of interest to investigate what influence special pathological changes had on the insensible perspiration rate. Some experiments were performed on patients suffering from diseases in which we know that the skin and the water metabolism are of special significance. Subjects suffering from nephritis, hyperthyreoidism, hypothyreodism, diabetes, pituitary diseases and rheumatic fever were examined. Not all cases are recorded, because it was not always possible to obtain all the necessary data. Only examples of cases in which complete data were obtained are included in Table V.

Nephritis.

The basal metabolic rate was sometimes increased in acute diffuse nephritis; it was nearly always decreased in the subacute cases. On the other hand, the rate was often increased in chronic diffuse nephritis, especially when heart symptoms were present. In all cases of nephritis the insensible perspiration—absolute and "relative"—was very low indeed, irrespective of the presence of œdema, and no correlation could be established between the degree of œdema and the insensible perspiration. As may be seen from the third and sixth columns of Table V, there is no parallelism between the deviation of the basal metabolic rate and the insensible perspiration, the latter being always much under the normal value predicted from Benedict and Benedict's chart, even when the basal metabolic rate is increased. This fact suggests once more that in cases of nephritis the condition of the capillaries—especially those of the skin—is altered.

Hyperthyreoidism.

There is a pronounced and concurrent increase in the basal metabolic rate and the insensible perspiration in hyperthyreoidism (see the third and sixth columns of Table V). Nevertheless, it is not possible to predict exact figures for one factor if the other factor has been measured. Although the highest figures for the insensible

TABLE IV.

Effect of Pyramidon: Subject E., Aged Seventeen Years, Body Surface 1·95 Square Metres.

Time of Experiment.	Average Skin Temperature. (Degrees Centigrade.)		Calories.			Insensible Perspiration. (Grammes per Hour.)		
	Before Cooling.	After Cooling.	Before Cooling.	After Cooling.	Difference. (Percentage.)	Before Cooling.	After Cooling.	Difference. (Percentage.)
Normal day	34	30·9	1,944	2,013	+3·5	35·6	26·4	−26
Pyramidon day	34	30·7	1,937	1,905	−1·7	33·6	25·4	−25

TABLE V.

Subject.	Age. (Years.)	Condition.	Calories.			Insensible Perspiration.			
			1 Normal.	2 Found.	3 Difference. (Percentage.)	4 Total Grammes per Hour.	5 Grammes per Hour per Square Metre.	6 Percentage Deviation of Predicted Value.[1]	7 Relative Insensible Perspiration.[2]
		Normal.			±5		(17–21) 19·5	0–10	(27–32) 30
L.	14	Acute diffuse nephritis.	1,300	1,509	+15	22·5	16·5	−17	20
B.	14	Acute nephritis after recovery.	1,463	1,563	+7	27·4	13·5	−10	24
Ba.	50	Subacute diffuse nephritis.	1,310	1,068	−18	19·3	11·7	−25	25
P.	27	Subacute diffuse nephritis with œdema.	1,620	1,601	0	27·0	15·7	−27	24
Lo.	52	Chronic diffuse nephritis.	1,532	1,835	+20	25·2	13·5	−24	19·3
H.	56	Chronic diffuse nephritis.	1,425	1,528	+7	23·0	13·5	−25	21
Ha.	47	Hyperthyreoidism.	1,224	1,598	+31	37·1	24·7	+54	32
G.	41	Hyperthyreoidism.	1,158	2,090	+72	32·0	22·5	+45	22
S.	27	Hyperthyreoidism.	1,360	2,071	+51	34·6	21·6	+21	23
J.	51	Hyperthyreoidism.	1,152	1,633	+41	31·3	21·4	+42	27
By.	40	Hypothyreoidism.	1,264	729	−42	14·6	9·4	−35	28
Ad.	20	*Diabetes mellitus.*	1,695	1,720	0	27·0	15·0	−30	22
Be.	18	*Diabetes mellitus.*	1,565	1,414	−10	22·4	13·6	−35	22
Ba.	53	*Diabetes mellitus.*	1,200	1,123	−6	19·0	12·6	−19	24
M.	25	Pituitary obesity.	1,890	1,956	+3	37·1	18·0	−18	26
R.	28	Fröhlich's syndrome.	1,405	1,400	0	20·7	12·7	−33	20
P.	14	Rheumatic fever.	1,601	1,594	0	47·6	29·0	+50	42 During the illness no visible sweating
			1,780	1,780	0	40·8	22·8	−3	32 After recovering.

[1] Benedict and Benedict's curve (Figure I) has been used to calculate from the normal basal metabolic rate corresponding value for insensible perspiration.
[2] The "relative" insensible perspiration is the percentage of the total heat loss due to insensible perspiration.

perspiration per square metre in the present series of experiments were encountered in these cases, it is noteworthy that the insensible perspiration is not increased to the extent that might have been supposed. It seems that there is a maximum value for insensible perspiration which cannot be passed without the onset of "sweating" (about 40% to 50% above the normal level as predicted by means of Benedict and Benedict's curve from the normal basal metabolic rate for the individual). The wet skin of patients suffering from thyreotoxicosis demonstrates this fact. Since in cases associated with an extremely augmented heat production the other means of loosing heat (sweating, radiation) have to take a higher share in the elimination of heat, the "relative" insensible perspiration cannot be increased.

In this limited series of cases of hyperthyreoidism, the parallelism between basal metabolic rate and insensible perspiration is not quite sufficient to suggest that the measurement of insensible perspiration can be a substitute for the measurement of the basal metabolic rate; but in any case it is valuable as a control method. On the other hand, it is quite possible that the determination of the insensible perspiration may be a more reliable method than the basal metabolic rate and this applies particularly to cases in which nervous excitement is present, since this by itself produces only slight variation in the insensible perspiration (provided that sweating and movement are excluded), although it produces pronounced variations in oxygen consumption.

Hypothyreoidism.

In hypothyreoidism the basal metabolic rate and the insensible perspiration are both extremely low and are decreased nearly in the same proportion. It therefore appears that the measurement of the insensible perspiration is a good method of controlling the basal metabolic rate in these cases.

Diabetes Mellitus.

In *diabetes mellitus* the basal metabolic rate is as a rule low. On the other hand, it was found that the insensible perspiration rate, both absolute and "relative", is very low, a finding which seems to be constant. The low value of the "relative" insensible perspiration (22%) indicates that the other main factor concerned in heat elimination, namely, radiation, must be relatively high in diabetes.

Fröhlich's Syndrome.

In Fröhlich's syndrome the basal metabolic rate was normal. In spite of this the insensible perspiration was low. In a case of "pituitary" obesity without genital dystrophy no pronounced deviation from the normal was observed.

Rheumatic Fever.

During the subacute stage of rheumatic fever the rate of insensible perspiration was extremely high. There was no visible sweating, but some "invisible" sweating could not be excluded in rheumatic fever, in view of the high values recorded. After recovery (during his stay in hospital the patient grew and gained in weight) normal figures for insensible perspiration were obtained. The basal metabolic rate was always normal.

SUMMARY.

The value of Benedict and Benedict's curve showing the fixed relation between basal metabolic rate and insensible perspiration was tested.

In 80% of all normal cases the figures for insensible perspiration did not differ from a range of —10% of the predicted values. In the remaining normal cases the figures for insensible perspiration were usually 10% to 20% lower than the predicted values. The figures are not reliable if the basal metabolic rate is higher than 1,800 calories per day or if the insensible perspiration rate is above 40 grammes per hour.

The figure for the total water loss per hour (without any correction), the so-called insensible perspiration, was found to be about 19·5 grammes per square metre; the "relative" insensible perspiration was up to 30%.

Reliable results in experiments upon insensible perspiration can be obtained only if the skin temperature is kept "normal" and constant, as slight cooling (a decrease in the skin temperature of about 2° or 3° C.) decreases the insensible perspiration rate by 15% to 20% and increases the oxygen consumption by 5%. The control of the skin temperature is therefore indispensable.

Young Australians seem to be more reactive to slight temperature changes than people living in moderate climates.

Day-time sleep decreases the insensible perspiration rate by about 15%.

Pyramidon has no effect on insensible perspiration, while it stops the normal increase of oxygen consumption that follows cooling.

Erythema brought about by ultra-violet irradiation decreases insensible perspiration greatly by means of a local effect.

The measurement of the insensible perspiration by means of the Sauter balance, which calls for little if any collaboration with the patient, is recommended for clinical purposes, because the results are reliable and reproducible. The method is a valuable supplement to and sometimes an improvement upon the determination of the basal metabolic rate as a clinical test.

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MILITARY MEDICAL EMERGENCIES.[1]

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A MEDICAL emergency is, I suppose, a condition which, if not adequately treated, will result in catastrophe. I imagine that most of you expect that tonight I shall talk to you about such medical catastrophes as apoplexy, coronary disease, abdominal perforations, gastric hæmorrhage, hæmoptysis, uræmia and epilepsy. These are, I suppose, the most common medical emergencies of civil life; but we do not, or should not, see them in military life. "Why?" you may ask; in the first place because of the age factor. Most of these diseases are diseases of at least middle life, and though older men in the later groups are being called up, still on the whole we are dealing with men outside the coronary block or uræmic age. Next, preliminary examination should exclude the possibly uræmic or tuberculous, although in the case of the ulcer subject and the epileptic we unfortunately must still rely on history. We may, of course, see these conditions closely mimicked. Thus a neurosis may equally simulate a stroke, a coronary block or an exophthalmic goitre. Still, I do not think that these cases should be regarded as medical catastrophes, unless, of course, the neurotic basis were not recognized and the condition were treated as organic. That would be a first-class medical disaster.

What, then, is a military medical emergency? One might define it, I think, as some unexpected threatened medical upset occurring in a soldier—the result of active service. The answer to this is, of course, that there ought to be no unexpected upsets; that they ought to have been foreseen and guarded against. Let me take some examples. In the South African War the typhoid epidemic of Bloemfontein was entirely unexpected—no measures had been taken against it; the result was a grave strategic dislocation.

[1] Lecture delivered to medical officers, Northern Command, on March 31, 1942.

The army, however, learned its lesson, and when a sound preventive measure turned up it was adopted, so that typhoid fever in the last war disappeared as a major military problem. Dysentery, on the other hand, in Gallipoli and France was not envisaged as a serious possibility, and until adequate measures were adopted, thousands of men were invalided and lost to the army.

Therefore it seems that the occurrence of a medical emergency on a large scale is a sign of a lack of foresight on the part of someone. Let me give an example. In a northern town it was recently reported that there were 2,000 cases of dengue fever. To my mind this constitutes a medical emergency of a grave nature. Dengue fever, while not fatal, is a gravely incapacitating disease, and its presence should cause as much anxiety as dysentery did in France in 1918. But dengue fever, we know, is carried by certain mosquitoes, and these mosquitoes are moderately easy to attack. The most important is the ordinary *Stegomyia fasciata* or *Aëdes ægypti*—easily recognized, not very alert or active, and above all a domestic breeder. Therefore dengue fever is controlled if we can keep down the *Aëdes* mosquitoes. This requires thought, organization, and above all education. Actually the work will be much heavier because of our "A.R.P." measures; fire-buckets, rain-filled trenches, dispersal arrangements, all help to make the task difficult for local authorities already embarrassed by shortage of manpower. However, a leading public health official tells that, in the words of Binks of Hezabad:

> I sought the City elders and my words were very plain—
> They flushed that four-foot drain head and it never choked again.

But the danger is there, and behind it looms a still greater one—that of yellow fever. The same mosquito carries dengue and yellow fever. At present we have no reservoir of infection in Australia; but just as it is probable that dengue fever has been reintroduced by the influx of infected individuals from regions where the disease is endemic, so we must remember that yellow fever is daily approaching the coast of East Africa, and that the East Africa coast is growing daily nearer to Australia.

What preparations are being made to face the possibility of such a serious medical emergency? No doubt many of my listeners will say: "Yes, but that is a job for the hygiene section of the Army; it does not concern the ordinary regimental medical officer." If yellow fever comes here, it will be his affair to send the sick patients to hospital. to notify the incident on Army Form OO and perhaps to carry out some mosquito control. But, my good fellow (as Demosthenes used to say), when any emergency has been successfully met early, that fact has been due to the skill and intuition of an individual medical officer, often a regimental medical officer.

In December, 1915, the Germans made the first use of phosgene gas in front of Ypres. A regimental medical officer, the late W. J. Adie, described the symptoms, especially the delayed collapse so characteristic of this gas's effect. When, twenty hours later, his report was read by the chairman of the Chemical Warfare Committee, the latter said at once: "Oh, they've started using phosgene at last." Appropriate defence measures were set in action and a great danger was averted. Hence my suggestion in regard to yellow fever would be: remember the possibility of its occurrence, learn the symptoms and meantime maintain vigorous mosquito control.

Let me give another instance of the regimental medical officer's forestalling a grave medical emergency. In 1916 Lieutenant Wiltshire, Royal Army Medical Corps, was in charge of a number of Serbian soldiers, among whom there occurred an outbreak of mild scurvy. His proper line of action, of course, would have been to report the matter at once and apply for an issue of oranges—a very unlikely article in Salonikan base depot medical stores at that time. Instead, Wiltshire remembered work carried out a few years before by Holst, and germinated haricot beans between moist blankets. Four ounces of the dry beans when germinated produced all the anti-scorbutic factor each man required daily, and the outbreak ceased.

To go back even further, it was a regimental medical officer—Surgeon Home of the Royal Dragoons—who first ordered drinking water to be boiled to prevent dysentery. This happened in Flanders in 1744.

Not only yellow fever, but plague from the ever pestilential East is another danger hanging over us today; smallpox is not negligible, while in the canefields of Queensland we have already present the dangerous and insidious infection of Weil's disease. I know that there are a dozen arguments against the possibility that these diseases will become a danger; but in epidemics, as in war, it is the unexpected that happens, and it is always the least likely form of attack for which we should prepare.

But to leave these hypothetical and only possible dangers, what actually have we now at hand? What are the gravest medical emergencies facing the services today? The first is the condition we are quite happily calling gastric or duodenal ulcer. I naturally cannot give you figures; but in the Royal Australian Air Force easily the largest proportion of men discharged on medical grounds are suffering from this condition. They usually give a consistent history of epigastric pain, frequently with finger-point location, often a hunger pain and less commonly a waking pain. Examination reveals a well-defined area of tenderness, often the rectus muscle is rigid, the biological chemist reports a climbing acid curve, and the radiologist reports an ulcer. Surely the picture is complete! One thing is needed; it is unfortunately not always looked for and still less often found—occult blood. Now some of these people give a history of ulcer before entering the services; many do not. The number was growing; I am not sure whether the approach to action is increasing or decreasing it. In the Royal Australian Air Force at least it is most common in the two most distasteful musterings (that is, occupations), those of messmen and guards. The Royal Australian Air Force is not alone in this trouble, and one has only to look at the *British Medical Journal* or *The Lancet* to see the many articles dealing with the problem.

Now there are two very interesting features of the condition as I have seen it here: (i) the complete absence of perforations, (ii) the rarity of hæmorrhage. It is because of the latter that I emphasized the importance of occult blood. Can you make any deduction from these facts? Another striking feature is the temperament of the sufferers. To use the colloquial word, they are "fed up"—"fed up" with their jobs, "fed up" with living in barracks, "fed up" with mass feeding. In almost every case they remarked that they wanted to stay in the Service, but that they wanted to live off the station, where they could get a better diet. Now if you consider the diet all these airmen were receiving, you will think it the answer to the dietitian's prayer. There was unlimited milk, there was meat, good and (in one station at least) well cooked; there were large quantities of green vegetables, ample fruit, wholemeal bread and few heavy carbohydrate puddings. When they leave the station to live at home, they have white bread and jam, milk, eggs, fish, masses of starchy puddings, little fruit, few green vegetables, less cheese, no wholemeal bread—and they flourish. Especially do they dread "acidy fruit and vegetables", though they are not at all sure what constitutes "acidity"; but tomatoes, pineapple and oranges upset them.

It has been suggested that in many of these men who had suffered previously from dyspepsia, dieting has set up a conditioned reflex. The voice of the medical adviser saying "you must not eat so-and-so, or such-and-such will happen" has been in their ears for years, and the result is that the prescribed food forms the first link in the chain of digestion. This prescribed diet stimulates the psychic flow which Pavlov's bell induced in his dogs, while the forbidden foods produce just such a depressing effect as that of the sprinkling of indol on the table. And it is surprising how many people are "on diet"—put there by themselves or by their doctor. Usually this dieting is done after no very adequate investigation. The patient retails his history; the medical adviser immediately diag-

noses a duodenal ulcer, prescribes a certain set diet and a powder to be taken after meals, and asks the patient to see him again some time. The patient, if he is neurotic, goes away quite happy; he has three things which are of the greatest importance to a neurotic patient: (i) a real disease with an impressive and well-known label; (ii) a form of treatment that is going to add greatly to his own importance and to the inconvenience of those about him; (iii) the prospect of never really being better. If he has an ulcer, he receives real benefit; if he has not, he receives mental ease.

What is the position when he comes into the army? First, he has no longer a family ready to form an audience for his symptoms; next, his own general importance is much diminished; and finally, food is placed before him containing all the things he has carefully avoided—especially those acidy foods, tomato and pineapple. What is the result? His stomach revolts, there is no psychic flow, food is forced onto an unwilling stomach, and he does have dyspepsia—a dyspepsia that all too closely mimics an ulcer. Add to this the other discomforts and tribulations of a soldier's life, and you will see that the neurotic soldier has ample material for a condition that will often with the utmost fidelity mimic a peptic ulcer. And what are you as regimental medical officers going to do about it?

Second in importance to the mock ulcer we have the mock coronary block and pseudo-anginal attack. In these conditions, I am glad to say, diagnosis is easier and the mimicry is less close. You know, for instance, that heart disasters like a coronary block are exceedingly rare in young people, that the pain of true angina is not under the heart, and that this pain comes on with the effort, not a little while afterwards; and moreover, angina seldom occurs without some accompanying physical sign. (It is true that at an Australian general hospital cases of coronary block have occurred in men aged under forty years—one aged thirty-seven and one thirty-four; but they were so remarkable as to call for special comment.) But still these people are unrecognized too often; they drift from regiment to hospital and from hospital to convalescent depot, and so acquire that obstinate anxiety state which is the despair of hospital staffs and repatriation departments.

Your regimental medical officer must stop these people early in the day and keep them away from the rear lines—that is, so long as the rear lines are safe places. (In England there were no safe places, and the number of neurotics in the Battle of Britain was negligible.) Literally, the duty of regimental medical officers may be summed up in the words "*sursum corda*". But let me warn you of two organic conditions which do produce "D.A.H.", the non-recognition of which may lead you into grave pitfalls. The first of these is the "influenza" group. We all know from experience that a bout of influenza leaves us languid, atonic and fit for nothing. Try as we will, we cannot drive our bodies to work with their old energy, and such an attempt in the soldier may easily induce "D.A.H.", both physical and emotional in type. So convalescence after these fevers should be a most carefully graded matter, with every mental stimulus to exertion; probably surfing is the best of these.

Tuberculosis is another problem. It is most difficult to distinguish between post-influenzal depression, incipient tuberculosis and neurotic effort syndrome. Fortunately there is at hand one very important weapon too seldom used—the theromometer. If such a patient after a brisk walk at 1730 hours does not show a rise of temperature (preferably rectal), then all the odds are against tuberculosis. If he has a rise in temperature in addition to exhaustion and distress, he is quite ill enough to be out of the front line in any case.

I believe that the most dangerous emergency which faces medical officers is the problem of morale. Many of the medical causes of invaliding, so far as I can see, have been frank hysteria, anxiety state, what you will. They manifest themselves in many ways, and it is because they so often mimic organic disease that they so greatly concern us. I said earlier that the mistaking of a functional condition for an organic one is a disaster; it is. Let a neurotic individual soldier or a civilian once acquire a label of organic disease and you will never do any good with him. He will retire behind his label and nothing will entice him from his dugout. Moreover, I cannot too strongly insist that the place for diagnosis of functional conditions is the front line. I know it is much easier to take the line of least resistance and say: "This man may have a gastric ulcer, he may have epilepsy, he may have a coronary block; I'll send him down to the base to be investigated." If you do, at once there is set up in that man's mind the doubt of your capacity to diagnose his condition, and any influence you may have had is gone. Two motives—one worthy, the other unworthy—are going to impel the average medical officer to evacuate the "not yet diagnosed" subject. The first—the worthy one—is that he may be unwittingly sending a sick man to his doom—compelling the incipient consumptive to work on and so extending his disease, driving the epileptic to further attacks and the man with a failing heart to death. The other—a less worthy motive—is the one which is the curse of State medicine—the unwillingness to assume responsibility. In any form of State medicine there is a temptation to avoid decisions, because one has to write something down on paper and put one's name to it. One, three, twenty years later that opinion may be dug up and thrown at our heads. For nearly twenty years I have been doing this work and I have learnt that the best course is to put down one's honest conviction in accordance with the evidence. It is always with a feeling of trepidation that one sees a file with a ten-year-old diagnosis come up again—right was it, or wrong? Looking back now, I can safely say that when I had the courage to give a decision in accordance with the evidence, I have seldom regretted it. When I have yielded to temptation and hedged, I have too often been shamed.

So the medical officer in most of these cases must take his courage in his hands and make a diagnosis. He will make mistakes; but he will do less harm than if he temporizes. The first thing in learning to make a diagnosis is to know your men. Not only will you be able to pick out the potential weakling, but your patients will talk freely to you about themselves and their diseases. It is from such talks more than from physical examination that you will make your diagnosis. By this, please do not imagine that I am advising you to some form of medical clairvoyance; I am not; but I am urging you to go back to the methods of our medical fathers, who made some very good diagnoses, for all that they had no X rays, no microscopes, no gastroscopes. The medical officer who knows his unit is like the family doctor who knows all his patients, their histories and their constitutions. He will know that Sergeant So-and-so will develop a cough every wet night and will stick to his job despite it, but that Private Such-an-one will likewise develop a cough, which he will at once present as the reason for a prolonged stay in a base hospital. The medical officer will know that his random visits to the kitchens will keep the cooks doing their best, and that fights for an extra blanket will lead to better sleeping and less grumbling among the troops.

Modern warfare is terrible enough in all conscience; if we are to leave men entirely to their own devices, they will suffer badly.

Shaken and unwell, he reeled back to camp. It was not far from midnight, and the exhausted man, his nerves all frayed, flung himself down across the mess-table to sleep.

That, you will say, was the beginning of an anxiety neurosis; the man would quite plainly never be any more use as a soldier. That was the reaction to his first battle of Arthur Wellesley, later Duke of Wellington. The actual fight we should consider little more than an encounter of patrols; but it shook Wellesley not only at the time, but even thirty years later.

He was not born, but made himself, the unmoving soldier of later years, and learning his lessons as they came, he learned some of them (since night attacks are a rough school of war) that night at Sultanpettah.

An embittered ex-sergeant wrote of the medical officers in the last war that "in their treatment of the sick almost all M.O.'s exhibited neither intelligence nor courtesy". If that standard holds, then the outlook is gloomy. I do not think it held in the last war, nor do I think it likely to hold in this. Fortunately the same sergeant also states that "in the line all M.O.'s worked like niggers, some of them heroically", and it may inspire you to remember that the only two bars to the Victoria Cross ever awarded went to two medical officers.

But troops are not always in the line; in fact, we may have long periods here, as has happened elsewhere, in which our troops will find time hanging heavily on their hands. Morale may then easily be lowered, as was that of the French Army through the winter of 1939-1940. There are many medical officers who can successfully go "over the top" with the battalion—even take the battered remnant of the battalion and lead it, as medical officers have done more than once. (To one such[1] did Haig say, when handing him his Military Cross: "If you had been a combatant officer, this would have been a V.C., but we cannot give a V.C. to a non-combatant who fights.") But the medical officer who by influence and good work can help sustain the morale of a battalion at rest is a much rarer person; yet it may be that it will be his work that will save the unit from the gravest of all military medical emergencies—I mean that sudden loss of morale which is the prelude to complete military disaster.

CLINICAL DIAGNOSES CONTRASTED WITH POST-MORTEM FINDINGS.

By J. B. CLELAND, M.D.,

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For nearly twenty years it has been the custom at the Royal Adelaide Hospital for the house surgeon to fill in what is known as a "request card" when he obtains permission to hold a post-mortem examination. On the "request card" is given the name of the patient concerned, with the dates of admission to hospital and death, together with a short clinical summary and the diagnosis that it is presumed would have been placed on the death certificate had there been no post-mortem examination. It has been my custom to place on the bottom of the card the chief post-mortem findings and then to mark the card as correct or reasonably so, partly correct, or wrong. These cards have then been filed away; but the need of old paper for pulping has led to my looking through them to see whether any information of interest or value could be obtained from them before they were sent away.

I have stopped analysing these at a total of 2,500 so as to have a convenient number to handle. Of these 2,500 cases, 1,697 (67·8%) were considered as correctly diagnosed before death, or at any rate reasonably so; 310 (12·4%) were considered partly correct; 362 (14·6%) were considered wrong; and in 131 (5·2%) no diagnosis was made, or the cause of death still remained obscure even after the post-mortem examination.

In a consideration of these figures it must be borne in mind that a large general hospital receives many patients *in extremis* or dying shortly after admission, before a proper examination can be made. It may be noted that in 89 at least of the 362 cases in which diagnosis was wrong, death occurred on the day of admission to hospital or on the following day—that is, in one-quarter of these cases. The diagnoses also are made by young practitioners who cannot as yet have much experience behind them to guide them in difficult cases. Taking the figures altogether, I think they must be considered creditable.

Certain pathological conditions have suggested themselves as worthy of further analysis and are given below.

[1] This officer was later transferred to a convalescent camp—he was a complete failure.

Malignant Growths.

Cancer, including malignant melanoma and sarcoma, was correctly diagnosed in 156 cases, to which may be added 10 in which the site was wrongly inferred—a total of 166. Carcinoma was present but not diagnosed in 27 cases, and was diagnosed or suspected but not present in 22. In addition, a patient who died from pneumococcal meningitis had a carcinoma of the *cervix uteri*, and one with carcinoma of the bladder died from massive pulmonary embolism. It must be remembered that these cases include patients admitted to hospital *in extremis* or in circumstances in which full examination is not possible. Eighty-six *per centum* at least of cases of carcinoma in mostly inaccessible parts were thus correctly diagnosed. Those cases in which this condition was not recognized are nearly offset by those in which it was suspected but was not present. There is, however, a slight balance, indicating that for statistical purposes the actual incidence of carcinoma is really a little higher than the figures of the Registrar-General suggest. It will be seen that in 27 out of the 2,500 cases analysed, or in about 1%, malignant growths were present but not suspected, and in 22 they were suspected but not present.

Cardiac Infarction.

Cardiac infarction (including two or three cases of fibrosis of the cardiac muscle) was correctly diagnosed in 48 cases, to which may be added two further cases in which the diagnosis was merely "congestive heart failure", a total of 50 out of the 1,697 correctly diagnosed cases.

Cardiac infarction was diagnosed but not present in nine cases. The causes of death in these were: lobar pneumonia (two), dissecting aneurysms of the aorta (two; one had ruptured into the pleura); rupture of an atheromatous aneurysm into the left pleura; glioma; chronic cerebral abscess; general peritonitis, probably from acute appendicitis; and collections of pus cells in the liver with grumous fluid in the pericardium.

Cardiac infarction was present but not diagnosed in nine cases. The diagnoses made were: hypostatic pneumonia; bronchopneumonia; asthma and bronchopneumonia; tuberculosis (there was some apical pulmonary tuberculosis); chronic nephritis with hemiplegia (cerebral softening was present); cerebral hæmorrhage (embolic softening was present); cerebral thrombosis, cough and cold with acute heart failure (sudden death from rupture of the heart); and old rheumatic heart disease with heart failure (the heart had ruptured).

Amongst the 21 cases in which no diagnosis was made and in which death occurred on the day of admission to hospital or the next day, there were one of infarction of the heart with rupture of a papillary muscle and one of thrombosis in the left coronary artery.

In those cases in which no diagnosis was given and in which death occurred more than forty-eight hours after admission to hospital, there were three cases of cardiac infarction, one of these patients being found dead in bed with rupture of the heart, and two cases in which death was attributed to coronary disease with obstruction or clot.

Summary.

Excluding cases of coronary disease associated with thromboses or obstruction but without obvious recent infarction, and also long-standing cases of fibrosis, there were just over 60 cases in which recent infarction was found at autopsy; in 48 of these, or approximately 80%, a correct diagnosis was made. In nine cases a wrong diagnosis was made, but for statistical purposes these are exactly offset by nine cases in which this condition was thought to be present and at the post-mortem examination death was attributed to other causes. Statistics of deaths from this condition, based on clinical diagnosis only, are probably thus substantially correct in spite of a considerable error in either direction.

Cerebral Vascular Accidents.

Among all the "request cards" there were 110 cases of ordinary cerebral (or cerebellar) hæmorrhage and 85 of cerebral softening. Cerebral hæmorrhage was diagnosed

correctly in 84 cases (including four cases of cerebellar hæmorrhage, three diagnosed as cerebral and one as "pontine(?)"; three cases of pontine hæmorrhage, two of which were diagnosed just as cerebral hæmorrhage; and two cases of cerebral hæmorrhage in leuchæmia correctly diagnosed).

In four cases the diagnosis was merely "cerebral vascular accident", in two it was hemiplegia only, in seven it was cerebral softening, in seven it was subarachnoid hæmorrhage—a total of 20 cases. Cerebral hæmorrhage was present but not diagnosed in four cases, and in two cases in which death occurred without a diagnosis on the day of admission to hospital or the next day, cerebral or cerebellar hæmorrhage was present. Thus, out of the 110 cases of cerebral hæmorrhage, in 104 the diagnosis was correct or reasonably so, in four it was wrong, and in two no diagnosis was given. Cerebral hæmorrhage was diagnosed but not present in 11 cases, in one of which post-mortem examination revealed an abscess of the brain as the cause of death.

Cerebral softening was diagnosed correctly in 52 cases. It was diagnosed as cerebral hæmorrhage in 22 cases, as meningo-vascular syphilis in one case; it was present but not noted in a patient who died from hæmorrhage from a duodenal ulcer, and it was diagnosed merely as a "cerebral vascular accident" in three cases; the total is thus 27. It was present but not diagnosed in six cases, in two of which uræmia was present. Thus out of the 85 cases of cerebral softening the diagnosis was correct or reasonably so in 79. Cerebral softening was diagnosed but not detected in nine cases, which included one of lysol poisoning, one of abscess of the brain and two of uræmia. Subarachnoid hæmorrhage was correctly diagnosed in seven cases, and present but diagnosed as cerebral hæmorrhage in three—total, 10. Subdural hæmorrhage was diagnosed correctly in three cases and diagnosed as cerebral hæmorrhage in two—total, five. An epidural hæmorrhage was diagnosed as "a vascular accident".

Summary.

From the foregoing results it will be seen that cerebral hæmorrhage and cerebral softening are usually correctly diagnosed, but that there are more cases of cerebral softening in which a diagnosis of cerebral hæmorrhage is made (22), than of cerebral hæmorrhage in which a diagnosis of cerebral softening is made (7).

Pneumococcal Peritonitis.

In three cases of pneumococcal peritonitis a wrong diagnosis was made, and in a fourth case no diagnosis was made.

CASE I: Post-mortem examination 30/34.—A woman, aged thirty-six years, died the day after admission to hospital. She had been ill for four days with abdominal pain, vomiting, diarrhœa, meteorism and headache. She had pronounced albuminuria, and pleurisy was present. She had had "nephritis" three years earlier. A diagnosis of "uræmia and ?" was made. The autopsy revealed generalized pneumococcal peritonitis and pleurisy at the bases of both lungs.

CASE II: Post-mortem examination 121/35.—A woman, aged twenty-six years, died three days after admission to hospital. She had been well till two days previously. She had vomited blood and passed flatus. A diagnosis of hæmatemesis and heart failure was made. The autopsy revealed pneumococcal peritonitis and also early cirrhosis of the liver with dilated varicose veins in the lower part of the œsophagus explaining the "hæmatemesis".

CASE III: Post-mortem examination 63/34.—A woman, aged twenty-four years, died two days after admission to hospital. The "request card" stated that she had had gastro-enteritis for four days and blood and mucus in the stools. The pulse was rapid and irregular, the temperature 104° F. She was suffering severely from shock. The autopsy revealed pneumococcal peritonitis.

CASE IV: Post-mortem examination 151/34.—A woman, aged forty-seven years, died two days after her admission to hospital with diarrhœa and vomiting. She complained of generalized abdominal soreness, but no rigidity was present. Her condition seemed much improved on the morning after her admission, but next day she suddenly vomited and then died. No diagnosis was made on the "request card". The autopsy revealed pneumococcal peritonitis.

Interesting Examples of Wrong Diagnoses.

The following are interesting, though not recent, examples of diagnoses corrected by post-mortem examination; they indicate how the statistical records of the causes of death, for the most part based merely on clinical information, are liable to err. It will be realized that in many instances the real cause of death, on the symptoms and signs available to the clinician, could not have been diagnosed. It should also be remembered that the same assemblage of clinical signs and symptoms may be produced by several different causes of different degrees of frequency, and that the diagnosis made should be the one of these occurring most frequently, the possibility of the condition being due to one of the less common causes being borne in mind.

Suppurating Hepatic Hydatid Cyst, Leakage, General Peritonitis.—The patient, a man, was admitted to hospital with diffuse tenderness and rigidity over the abdomen. Operation revealed free turbid fluid with lymph flakes in the peritoneal cavity, thought to be due probably to a ruptured gastric ulcer.

Ruptured Duodenal Ulcer, General Peritonitis.—The patient, a man, gave a four-day history of diarrhœa and pain, and was admitted to hospital in a condition of severe shock, gradually becoming weaker. Dysentery was suspected.

Leakage of a Saccular Aneurysm of the Abdominal Aorta, Extravasation into the Pelvis of the Kidney.—The patient, a man, complained of pain in the left loin of one week's duration. Tenderness on palpation and dulness to percussion were present in the left loin. He had no urinary symptoms and no pus in the urine, though there were numerous casts. He was thought to be suffering from perinephritis and uræmia.

Bronchopneumonia with Lobar Spread.—The patient, a woman, vomited one day and then lapsed into coma and did not regain consciousness. The diagnosis was "uræmia (?)".

Fibrosing Pulmonary Tuberculosis with Pleural Effusion.—The patient, a man, was considered to be suffering from heart failure of obscure origin. He had purpuric hæmorrhages on the forearms and an enlarged liver, thought to be cirrhotic.

General Peritonitis from Perforation in the Hepatic Flexure, Cause Unknown.—The patient, a man, was admitted to hospital with cardiac asthma, and the Wassermann test produced a positive reaction. His death was believed to be due to a failing heart from high blood pressure.

Extensive Silicosis.—The patient, a man, died whilst being examined, it was thought from acute pulmonary œdema.

Acute Infective Arthritis; Diffuse Purulent Infiltration of the Thyreoid Gland.—The patient, a man, had swelling and redness of one great toe, followed by swelling of both wrists, the left ankle and the left knee. His voice was hoarse. He had been ill for five days. He was considered to be suffering from bronchopneumonia and probably gout.

Summary.

1. In 2,500 cases in which autopsy was performed, the diagnosis was right or reasonably so in 67·8% and partly right in 12·4%, a total of 80·2%. In 14·6% the diagnosis was wrong and in 5·2% no diagnosis was made or the nature of the case remained obscure even after autopsy. These percentages probably can be applied in general to the returns on which the Registrar-General bases his statistics.

2. In malignant disease the number of cases of cancer missed are nearly but not quite counterbalanced by those in which it was suspected but not present.

3. In cardiac infarction, in 80% of the cases a correct diagnosis is made. The instances in which this condition is present but not diagnosed are counterbalanced by those in which it is diagnosed but not present.

4. Cerebral hæmorrhage and cerebral softening are usually correctly diagnosed, but more cases of softening are diagnosed as hæmorrhage than the reverse.

5. Primary pneumococcal peritonitis is a condition which is apt to be wrongly diagnosed.

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THE NEW SOUTH WALES POST-GRADUATE COMMITTEE IN MEDICINE AND ITS ACTIVITIES.

MANY non-medical people need to be reminded that medical education does not end with the conferring of a university degree and the assumption of the courtesy title of "Doctor". There may be some cynics who would say that certain holders of the honourable title of "Doctor" stand in need of the same reminder. Be that as it may, non-medical members of hospital boards should know that when an institution becomes known as a teaching hospital it has gained a distinction that sets it apart from others not so blessed. There are many reasons why this should be, and chief among them is that clinical work, the actual treatment of the patients, is carried out in a teaching hospital in the presence of the keenest critics who have a habit of asking searching and sometimes embarrassing questions. When the students who sit in critical judgement on their teachers are graduates, the standard of teaching and therapy is high and those who are responsible for the safety of the patient have little to fear.

In every State in the Commonwealth post-graduate study in medicine is in the hands of a committee to which medical practitioners turn when they are in need either of general refresher courses or of special courses of study. Australia has not yet reached the point at which every registered practitioner is required by law to engage in organized post-graduate study at stated intervals of time. That point may or may not be reached, but until it is reached the requirements of voluntary post-graduate students will be met by the post-graduate committees in the several States. The outbreak of war and the transference of more and more practitioners from civil to naval, military and air force medicine have affected the activities of these committees. Certain of the activities have ceased, not because study is any less necessary in wartime, but because a large number of practitioners are serving with the armed forces and those left behind are doing much more than is usually regarded as the work of one practitioner. But in spite of the absence of so many practitioners on active service and in spite of the heavy burden that is being carried by those remaining at their posts in civil practice, all organized post-graduate study should not be allowed to cease or the spirit actuating it to be quenched. Our object in drawing attention to this subject is to put on record the achievements of the New South Wales Post-Graduate Committee in Medicine during the past twelve months, to let practitioners know the present difficulties and to urge a continuance of effort wherever and whenever activity may be possible.

In its report to the Senate of the University of Sydney for the year ended December 31, 1941, the New South Wales Post-Graduate Committee in Medicine states that conditions created by the war disturbed the normal routine and made the committee curtail many of its activities. In spite of this we find that a course was held for candidates who intended to sit for the examination for membership of the Royal Australasian College of Physicians, a week-end course in electrocardiography was held and there were two week-end courses in medicine. On the surgical side a special course suitable for candidates for Part II of the examination for the degree of Master of Surgery was held. There was one week-end course in surgery and another had to be cancelled; a special course in urology was held and there were two courses in advanced anæsthesia. Conditions were not favourable for the holding of the usual courses in obstetrics, but facilities were made available for post-graduate students to attend the clinics at the Women's Hospital and the Royal Hospital for Women. A course in pædiatrics was held and demonstrations of the *fundus oculi* were given. A course in practical pathology attracted a number of students and medical officers from units of the armed forces also attended. Two courses were held at country centres. Special lectures were given in connexion with the national emergency as follows: a series of lectures on the medical aspects of air raids; a series of lectures on war surgery, attended by medical officers of the Australian Imperial Force; lecture-demonstrations on the preparation of hospitals for air raids; a special course of lectures for members of the Voluntary Aid Detachment who had already had practical hospital experience. Library seminars and clinico-pathological conferences were held every month; these extremely valuable meetings were fairly well attended, though towards the end of the year stress of the war situation made the attendances smaller. One of the most interesting features of the work is that arrangements were made for a number of post-graduate students to receive individual tuition in such subjects as anæsthesia, dermatology, gynæcology, leprosy, general medicine, obstetrics, pædiatrics, pathology, surgery, treatment of varicose veins and venereal diseases. These facilities, which in view of the difficult days of the present must be regarded as very creditable, have been made available to the medical profession by the Post-Graduate Committee because of the close liaison between it and the Prince Henry Hospital Post-Graduate School. The nature of this liaison is shown by the fact that the Chairman of the Post-Graduate Committee is the committee's representative

on the Board of Directors of the Prince Henry Hospital. Again it is the Senate of the University of Sydney which appointed the part-time post-graduate lecturers in medicine and surgery on the recommendation of the Post-Graduate Committee and the Board of the Prince Henry Hospital, and the board has appointed them respectively Directors of the Post-Graduate Units of Medicine and Surgery. The position of the Post-Graduate Lecturer in Pathology is somewhat different, for it is a whole-time position. But here the same liaison obtains for the university appointed the post-graduate lecturer and the hospital board appointed him Director of the Pathology Department.

From now onwards matters will be much more difficult for the Post-Graduate Committee. The Prince Henry Hospital has by order of the authorities been evacuated; the only beds at present occupied are those for patients with infectious disease. The training of Fellows in medicine and surgery has, of course, ceased and all post-graduate teaching at the Prince Henry Hospital has temporarily come to an end. The difficulties have been somewhat simplified by the generous resignation of office by the part-time post-graduate lecturers in medicine and surgery—and we are informed that the resignations have been accepted for the duration of the war only. It would be quite simple for the committee, having neither special hospital nor post-graduate lecturers appointed by the university, to sit and fold its hands in idleness until the war was over. But this will not be done. It will still be possible for special courses of instruction to be arranged for individuals, and we hope that special courses for groups of persons will still be arranged at suitable times. In this regard it will not do to wait till opportunity offers; the opportunity must be sought. The Post-Graduate Committee in its report acknowledges help it has received from many hospitals; there is no doubt that every one of these hospitals will be ready to help again if it is asked for help. But help is much more effective if it is offered and not sought. At the outset of this discussion we tried to show that hospitals given over to post-graduate teaching attained a position of dignity and importance. The hospitals in and around Sydney which are not occupied as teaching hospitals for undergraduates have now a chance of improving their status and of helping to make the practice of medicine more effective—and this after all is one of the objects of their existence. In any case the search for knowledge among practitioners and the spirit that actuates it must not be allowed to become extinct.

Current Comment.

CROSS-INFECTION OF WOUNDS IN HOSPITAL WARDS.

MEDICAL men of today have been born into the aseptic tradition. They know what the ravages of sepsis in a hospital ward can do, but very few have seen its worst results. Medical practitioners also know something of the investigations carried out from time to time into the way in which infection spreads in a surgical ward. Some have doubtless tried and are still trying to put into practice the lessons drawn from this work; but the matter is very complex and only by the most meticulous attention to detail can worthwhile results be achieved. As a matter of fact the results are far from perfect, and careful clinical and experimental observations are still being made with the object of defeating the ravages of sepsis. Though a practitioner charged with the care of surgical patients be ever so careful, it is simple to overlook some detail or to look on a step in technique as not really essential. Some practitioners may not have bothered to follow the work on this subject, which, it should be remarked, is becoming of growing importance in view of war conditions. In the circumstances a useful purpose will be served if the attention of medical practitioners is directed to some recent discussions and investigations on cross-infection of wounds. First of all we intend to describe some investigations by R. E. Willits and R. Hare, of Toronto, Canada,[1] and then to refer to a discussion on the subject which was staged at a meeting of the Section of Surgery of the Royal Society of Medicine in November last.[2,3]

Willits and Hare state that it is too readily assumed that septic infection of accidental wounds is inevitable by reason of the organisms which enter them at the time of infliction, and that infection of clean operation wounds is invariably due to a breakdown in the technique of the theatre. Their investigations were carried out in the ward. They made surveys of the skin of patients and of their bedding, and also made observations on air contamination. A total of fourteen patients was studied. Only three of these suffered from infected accidental wounds. The remainder had a purulent discharge due to an infected blister or ulcers or following drainage of some deep infection. It was found first of all that the skin of areas some distance from the wound might be contaminated by organisms of the same type as those present in the wound. In eight of fourteen cases (one patient was the subject of two different investigations) in which this was noted, the organisms were also present in the naso-pharynx. Thus some of the organisms on the skin might have come from the naso-pharynx. Willits and Hare point out that previous investigations have shown that contamination of wide areas of skin of naso-pharyngeal carriers may occur when there is no infected wound present as a complication. On the other hand, they found that in three instances contamination of the skin of areas some distance from the wound occurred when the patients were not naso-pharyngeal carriers. Clearly there is a danger that a nurse or surgeon handling these patients may unwittingly transfer organisms to other patients. In regard to organisms in the naso-pharynx, Willits and Hare promise information in a paper soon to be published which will show that a higher proportion of patients with infected accidental wounds are carriers than is usually supposed. Hæmolytic streptococci were found on the bedclothes—on the lower sheet in ten out of fourteen cases, on the bedspread in seven cases and on the upper sheet and pillow in five each. The blankets were examined in only three instances, and in these they were not infected. The conclusion is that the bedclothes may be contaminated by hæmolytic streptococci even when wounds are small and have little or no discharge; this occurs as much in the case of patients with the organisms in the naso-pharynx as in that of those without. The whole of the contents of the bed must thus be looked on as a potential source of organisms for the transfer of infection. Anyone who handles any part of the patient or his bed may contaminate his hands or any instrument which he uses.

Willits and Hare also made investigations of air currents. Though they cannot say that air pollution invariably occurs, they find that it occurs sufficiently often to make these patients a potential menace to their neighbours. They also report some ingenious experiments which showed that for release of infected material to occur from dressings, drying and some form of movement (movement of the dressing or of the surrounding air) were necessary. It was found in addition that quiet and careful bed-making released almost as many organisms into the atmosphere as the usual violent methods.

[1] *The Canadian Medical Association Journal*, December, 1941.
[2] *Proceedings of the Royal Society of Medicine*, December, 1941.
[3] *The Lancet*, November 15, 1941.

The Royal Society of Medicine discussion was opened by Professor A. A. Miles, who said that clean wounds were infected from two main reservoirs. One was the upper respiratory tract, and cross-infection in a community of nurses, patients and doctors was probably constant. He went on to say that the majority of carriers had no sign of infection and they might be as prolific a source of *Streptococcus pyogenes* as a person with acute tonsillitis. Sometimes there was an intermediate self-infection of a cut finger or a badly manicured nail—"the hand ceases to be an entrepreneur and goes into production as a manufacturer of streptococci". The other main reservoir mentioned by Miles and described by him as probably the most fruitful source of hospital infection is the streptococcal wound. This is the type of wound discussed by Willits and Hare. Miles states that when a patient with such a wound is attended the disturbance of infected dressings, pillow-cases, sheets and nightgowns showers streptococcal dust into the air. This takes place from material that is visibly soiled with discharge and also from apparently unsoiled material. The dissemination of infected material is very complete, for, as Miles points out, fine flakes of dry streptococcal exudate are carried by hands, clothing and instruments to other patients or are handed from one patient to another on books and newspapers. Ward utensils that are in common use may carry the cocci, for example the ordinary wash bowl. Miles also referred to the possibility that infected particles may be deposited from the air directly onto the wound. Miles laid particular emphasis on the prophylaxis and technique in the dressing of wounds. This was previously described by him in the *British Medical Journal* of September 13, 1941. In this paper will be found the detailed instructions to be issued to ward personnel in regard to the dressing of wounds. We should like to reproduce these instructions in full, but this is impossible. Readers would be well advised to consider these instructions and go over them with the technical and nursing staffs of any operating theatres in which they work. According to the technique described dressings are done by teams in which each member has his or her specially defined duty. The team consists of a trolly assistant on the one hand and a dresser and a part-time or whole-time assistant on the other. The trolly assistant supplies the sterile and clean materials to the dresser—all sterile material is handled by forceps and the trolly assistant never touches the patient or any soiled bandages or dressings removed from him or his bed. The chief concern of the dresser and his assistant is to treat each bed as a separate unit; all possibly infected material is carefully placed in special buckets; everything handled by the dressers during the dressing is sterilized and the two workers wash before going to the next dressing. The dresser may use a "no-touch" technique with dry hands or dress the wound in dry, sterile rubber gloves; dressing with "scrubbed-up" wet hands should be avoided. The precautions to be adopted include masking, the closing of windows and doors *et cetera*. Some figures quoted by Miles are interesting. Two periods are compared. During the first an ordinary dressing technique was adopted; there were ten cross-infections among 32 air raid casualties and two after 46 operations. During the second period in which the special team technique was used there was one cross-infection after 46 air raid casualties and there were none after 49 operations.

Professor J. Paterson Ross made an important point when he said that it was essential to review carefully the methods adopted for sterilizing instruments, utensils and all materials used in doing dressings. He insisted that before the war there had been a great decline in the standard of aseptic technique—what was advocated was nothing new, but really a revival of a proper interest in asepsis; there was also nothing elaborate in shutting windows, wearing masks and boiling instruments.

Colonel Leonard Colebrook told a story of multiple infections that followed the admission to hospital of a patient with severe burns which became infected with a streptococcus resistant to sulphanilamide. He also said that cross-infection was a bacteriological problem and should be dealt with by bacteriologists. He favoured the idea of an "asepsis officer" who would succeed in getting the facts over to students and nurses. If it was left to the surgeons there would be spurts of enthusiasm followed by lapses.

Sir James Walton remarked that if war came into Great Britain gross infections would be seen, and he thought that every medical officer should learn to adapt himself to what he could get. It was possible to dress wounds entirely without touching them and thus without washing between cases. A man could not do forty dressings, washing between each; but he could go round with forceps, using a separate packet of dressings and freshly sterilized instruments for each patient, and never touch a wound.

The investigations that have been described and the opinions that have been quoted are valuable first of all because they show where danger lies, and secondly because they give particulars of what may be described as an ideal method of carrying out surgical dressings in the present state of knowledge. The ideal is not always attainable, and Walton's contention that every medical officer should learn to adapt himself to what is available is obviously correct. The danger, of course, is that what is available may by reason of a "lapse" be thought sufficient even when opportunities to use ideal methods occur later on. Discussions such as those reported serve to awaken a "spirit of enthusiasm" and should take place from time to time. The whole ground has not been covered in the present instance; to fill in the gaps will prove to be a useful exercise and a further stimulus to enthusiasm.

CHANCRE OF THE GUM.

EXTRAGENITAL chancres are rare, and of these lesions chancre of the gum is one of the rarest. Sequeira states that less than 5% of chancres are extragenital, and in most text-books whose authors refer to extragenital chancres the gum is not mentioned as a site. The most important consideration in regard to extragenital chancres is diagnosis, and diagnosis of a primary syphilitic lesion in the buccal cavity is complicated by the fact that spirochætes other than *Spirochæta pallida* are sometimes found in the mouth. A report which brings out one or two interesting features has been made recently by J. F. Sadusk, junior, and B. G. Anderson.[1] The lesion which is described by them and is illustrated by an excellently reproduced coloured plate, occurred in a nineteen-year-old Negress. It appeared as a sore red spot on the gum above the left lateral incisor tooth; the spot soon increased in size and became painful, bleeding when the patient cleaned her teeth. There was an enlarged and tender cervical gland at the angle of the jaw. The patient was sent to the dental clinic and afterwards back to the medical clinic again. The lesion at this stage is described as being situated on the labial gingiva overlying the alveolar bone covering the roots of the two left maxillary incisors as well as portions of the left cuspid and right first incisor, extending down over portions of the crowns of these teeth. It was about one centimetre by two centimetres in diameter and was uniformly elevated about three millimetres throughout, with a clearly defined border. In contrast to the normal gum, the lesion was non-pigmented, spongy and had a bright deep red colour. It was at this stage that chancre was suspected. Serological tests for syphilis gave positive reactions, but the spirochæte was not found on dark field examination. A Negro with whom the patient had had intercourse was found to be infected; his lesions and those of the patient's sister yielded spirochætes which were seen in the dark field. The authors point out that chancres of the gum may be abrasive or erosive or else ulcerative; they must be differentiated from hypertrophic gingivitis and epulis.

[1] *The American Journal of the Medical Sciences*, March, 1942.

Abstracts from Medical Literature.

THERAPEUTICS.

The Treatment of Angina Pectoris with Testosterone Propionate.

MAURICE A. LESSER (*The New England Journal of Medicine*, January 8, 1942) records the results obtained in the treatment of twenty men and four women, varying in age from forty to seventy-seven years, who were given testosterone propionate for *angina pectoris*. Each of the patients selected experienced at least one anginal attack a day. They were given no medication except nitroglycerine to relieve an attack, were kept ambulatory, and were encouraged to perform their usual duties. Twenty-five milligrammes of testosterone propionate dissolved in one cubic centimetre of sesame oil ("Perandren") were given intramuscularly every second to fifth day, depending on the frequency and severity of cardiac pain and the response to treatment; a total of between five and twenty-five injections was given, with an average of eleven injections. No untoward reactions were noted. All patients in this series manifested improvement under testosterone propionate treatment, but the improvement was much greater in the men than in the women. There was no clinical improvement in the condition of seven patients who received sterile sesame oil, but improvement occurred when testosterone propionate was used. The action of testosterone propionate is not instantaneous and will not relieve an acute attack. The majority of patients show clinical improvement only after several injections, but the relief obtained persisted for from two to twelve months after treatment was discontinued. Severity and duration of attacks of *angina pectoris* were diminished, and these patients were able to increase their physical activities to a considerable degree without precipitating attacks. Fluoroscopic examinations, serial kymograms and electrocardiograms showed no uniform changes resulting from this therapy. In the majority of patients there was a lowering of blood pressure during the course of testosterone propionate therapy. The authors believe that testosterone propionate may prove to be a valuable drug in the treatment of *angina pectoris* and that further investigation is warranted.

Hæmatmesis.

J. T. E. NICHOLSON AND T. G. MILLER (*The American Journal of Digestive Diseases*, December, 1941) describe the results of prompt feeding for bleeding gastric and duodenal ulcer. The authors used this treatment in 32 consecutive cases. They point out that the so-called Meulengracht diet does not necessarily contain meat. At first the authors gave the full Meulengracht diet, but soon learned that the patients did better if started on a soft bland diet of milk and cream, cereals, eggs, puréed vegetables, cooked fruit and breadstuffs. Six or more feedings a day were given. In some cases only the gelatin mixture of Andresen was given to begin with. Water was given by mouth as desired. Phenobarbital or morphine was given for restlessness, and magnesium trisilicate or aluminium hydroxide to relieve epigastric distress. No hæmatinics were given. Blood transfusion, 250 to 500 cubic centimetres, was given in 13 cases. Only one patient in this series died. This man was found to have a perforated duodenal ulcer. Another patient continued to pass blood *per rectum* for a week. He was operated upon and a freshly healed duodenal ulcer was excised, though no blood was found in the stomach or duodenum. A third patient was operated upon, but no ulcer was found and his bleeding ceased spontaneously. In 29 cases hæmorrhage ceased within one to four days, but in 10 cases it persisted in smaller amounts for a longer time; in one duodenal ulcer case the bleeding persisted for twenty-three days. In two cases bleeding recurred on the seventh and twenty-first days respectively. On this full feeding treatment the patients were less nauseated and rarely vomited; persistence with the diet overcame nausea and vomiting, the morale was improved, anxiety was lessened and convalescence shortened. The authors analysed 1,396 cases in the literature in which treatment of hæmorrhage was by prompt feeding. The mortality was only 3%, as compared with 6% to 8% by older methods. They state that morphine is contraindicated, as it lessens gastric and duodenal tone and promotes nausea and vomiting.

Tetany.

F. E. HARDING (*The Journal of Laboratory and Clinical Medicine*, January, 1942) describes the use of dihydrotachysterol in parathyreoprivic tetany. This drug will raise blood calcium when given orally, whereas parathyreoid extract must be given hypodermically. With dihydrotachysterol the blood calcium rises after forty-eight hours, reaches a maximum in one week and declines in one to three weeks after its use is discontinued. Excess of the drug causes asthenia, nausea, vomiting, headache, stupor, thirst and skin rash. This drug does not mobilize calcium by removing it from the bones. The dose of the drug necessary in tetany varies between one cubic centimetre given twice a week and one cubic centimetre given every day. A case is reported in which cramps in arms and hands and extreme nervousness and prostration followed thyreoidectomy. The cramps were relieved by injection of parathyreoid extract daily or every other day. The blood calcium content was only 7·5 milligrammes *per centum*. Dihydrotachysterol was then given in doses of one cubic centimetre daily, later reduced to one cubic centimetre twice a week. The patient's condition improved, the blood calcium content rose to 12 milligrammes, but the symptoms continued.

Soldier's Irritable Heart.

PAUL D. WHITE (*The Journal of the American Medical Association*, January 24, 1942) deals briefly with the soldier's irritable heart. The condition has been described as neuro-circulatory asthenia and effort syndrome. True irritability of the heart as indicated by extrasystoles or paroxysmal tachycardia is not associated with the above condition. Nervous tachycardia is not usually associated with extrasystoles. Soldier's irritable heart is commonly associated with a psychoneurosis; there is apparently a very close relationship between neuro-circulatory asthenia and neurasthenia. In England it has been found wise to treat most patients suffering from neuro-circulatory asthenia as though they are suffering from ordinary fatigue and not to refer them to the psychiatrist or cardiologist; this is done in order to avoid over-emphasis on either the heart or the mental state.

Lobar Pneumonia.

A. LEVITT, H. T. SCHWEITZER AND K. GOLDSTEIN (*The Journal of Laboratory and Clinical Medicine*, January, 1942) recorded the results of the treatment of pneumonia by means of sodium sulphapyridine given intravenously. The diagnosis of pneumonia was corroborated by X-ray examination. A liquid diet of at least three litres a day was given; it was made up of fruit juices and milk. If fluid was not taken by mouth in sufficient quantity saline solution and glucose were administered subcutaneously. Sodium sulphapyridine monohydrate was given slowly by the intravenous route in 5% solution, four grammes of the powder dissolved in 80 cubic centimetres of distilled water. Eighty cubic centimetres were given daily and the blood was examined for sulphapyridine content frequently during the twenty-four hours. After one injection the blood level was eight milligrammes *per centum* in ten minutes and five milligrammes *per centum* at the end of twenty-four hours, whereas when the drug (sulphapyridine) was given orally, two grammes on admission to hospital followed by one gramme every six hours until three days after the temperature became normal, the blood level was 3·93 milligrammes *per centum*. With intravenous therapy, in this series two patients died; one was a senile patient and one died from toxic hepatitis. Results otherwise were similar to those obtained after oral administration; toxic symptoms, nausea, vomiting, cyanosis and headache were rather more severe.

Heparin in Subacute Bacterial Endocarditis.

JAY MCLEAN *et alii* (*The Journal of the American Medical Association*, November 29, 1941) discuss the use of heparin in 67 cases of subacute bacterial endocarditis, and conclude that the results do not warrant further trial of heparin treatment according to the plan used at present.

NEUROLOGY AND PSYCHIATRY.

A Study of Migraine.

DOUBT has been expressed as to whether migraine exists as a distinct pathological entity; and C. T. Gayley (*The Journal of Nervous and Mental Disease*, November, 1941) brings forward evidence to show that it is a psycho-somatic manifestation. In his opinion migraine may be regarded as a hyperfunctioning of the effort syndrome. He claims that an intense nervous strain stimulates and brings into action the sympathetico-adrenal mechanism, and by this preparation of the body to meet emergency by exertion, the metabolic rate is increased. The emergency is a false alarm. No exertion is required; but in the delayed action of decreasing metabolism intermediate oxidation products are formed which cannot be quickly eliminated. The kidneys have been thrown into a state of partial stasis by the sympathetico-adrenal mechanism, and therefore cannot effectively eliminate waste products which

accumulate in the blood stream and cause headache. The cessation of the migrainous attack corresponds with increased kidney elimination and polyuria. The C/N quotient in urine samples was found to be increased during the headache. In periods free from mental strain there is no migraine. The author assumes the inheritance of an imbalance in the sympathetico-adrenal mechanism.

Familial Periodic Paralysis Associated with Exophthalmic Goitre.

REFERRING to the literature of familial periodic paralysis, Alice G. Hilderbrand and Edwin J. Kepler (*The Journal of Nervous and Mental Disease*, December, 1941) state that five out of the sixteen cases seen at the Mayo Clinic were associated with exophthalmic goitre and that thyreoidectomy resulted in the relief of both conditions in all five instances. They quote a further case, that of a Pole, aged twenty-nine years, whose brother was similarly afflicted. It was found that his attacks could be precipitated by various means devised to depress the concentration of serum potassium, although the administration of large quantities of ionic potassium would not abort an attack. Recovery occurred after a subtotal thyreoidectomy and then it was observed that it was impossible to precipitate an attack of paralysis by depressing the serum potassium. The author believes that exophthalmic goitre is therefore a precipitating agent of this type of paralytic syndrome.

Intravenous Injection of Solution of Zinc-Insulin Crystals in the Treatment of Mental Diseases.

IN the hope of avoiding allergic reactions, Phillip Polatin, Hyman Spotnitz and Angelo J. Raffaele (*The Journal of Nervous and Mental Disease*, January, 1942) describe the treatment of psychotic states by the use of zinc-insulin. The technique of the intravenous administration of zinc-insulin crystals is given. Ten patients were treated, nine of whom were suffering from schizophrenia. The condition of three was improved and that of seven remained unchanged. Intramuscular injection of thiamin chloride had no influence on the rapidity of the development of the hypoglycæmic shock. Some untoward symptoms, such as irregularities of pulse rate and respiration and a tendency to convulsive seizure, were noted. Otherwise the authors found no advantage in the use of zinc-insulin over the ordinary method of treatment with unmodified insulin.

Psychoneurosis in a Hospital for Mental Disease.

TWO HUNDRED unselected cases of psychoneurosis occurring in patients of both sexes have been analysed and followed up by Clifford B. Farr and Genevieve M. Stewart (*The Journal of Nervous and Mental Disease*, February, 1942) in an attempt to gauge the prognosis in those cases in which commitment to a mental hospital had seemed necessary. The incidence of such disorders appeared commoner in women; the rate of recovery was greater, but relapses were more frequent than in men. This investigation showed that psychoneurotic patients, despite the popular belief, do at times become psychotic. Psychoses subsequently developed in 17% of patients who had a diagnosis of hysteria, in 19% of those with compulsion neuroses, in 12% of neurasthenics and in 8% of patients with anxiety states. Evidence contrary to the accepted belief that neurotics do not commit suicide was also brought out in this investigation. Of the 200 patients studied, 8% sooner or later committed suicide; and in a further 18% suicidal attempts were either made or mooted.

Exhibitionism.

AFTER giving details of five cases of exhibitionism and referring to the standard literature, N. K. Rickles (*The Journal of Nervous and Mental Disease*, January, 1942) submits the following classification: (*a*) expomania, exhibitionism due to compulsion neurosis; (*b*) depraved exhibitionism, including all cases in which the exhibitionist has carnal knowledge as his intent; (*c*) psychosis, which may be due to general paralysis, epilepsy, senility or alcoholism. The group of compulsive neurotics show a definite personality make-up; they are shy, retiring, over-conscientious and usually well educated. As a rule they are found not to have been able to make a satisfactory hetero-sexual adjustment and to have continued the practice of masturbation into adult life. Simple psychotherapy, in the author's experience, proved beneficial in these cases and the compulsion to exhibitionism was either greatly lessened or completely eradicated.

Familial Cortical Cerebellar Atrophy.

BRUCE HALL, K. B. NOAD AND O. LATHAM (*Brain*, Volume LXIV, Parts 2 and 3, 1941) describe a familial disorder which they believe to be identical with that set out by Gordon Holmes as familial cortical cerebellar atrophy. Several generations of this family were studied. A post-mortem examination on one of the family who died revealed chronic cortical parenchymatous atrophy of the Purkinje cells and secondary involvement of the olivary neurones. Ataxia of the lower limbs and speech difficulties were among the earliest clinical findings. The age at which symptoms became manifest was very close in several members. Nystagmus developed during the progress of the disease. Sensory disturbances were not found. The disease tended to progress to a stage of complete helplessness, but had no apparent effect upon longevity.

Neurosis Simulating Organic Disorder.

G. T. COOK AND WILLIAM SARGANT (*The Lancet*, January 10, 1942) deplore the fact that the diagnosis of neurotic states is generally accomplished by the exclusion of organic disease. Attention, they say, has been focused on the somatic aspect to the patient's detriment. They illuminate these points by an analysis of fifty patients in a base hospital. Forty-two of these patients gave a history of neurotic predisposition or severe mental disorder in one or more relatives; a high proportion were of low intelligence. The complaints included weakness, muscular pains and a variety of visceral symptoms. The patients were found to be suffering from well-known psychoneurotic disorders, and yet 19 of them had between them been examined by X rays 30 times, the examinations including 12 barium meals, two barium enemata and two excretion pyelograms. The authors claim that the earlier employment of neuropsychiatric investigation would save both time and money and prevent wrong diagnoses and the carrying out of much unnecessary treatment.

The "Coramine" Treatment of the "Reversible" Psychoses.

SEARCHING for a substitute for "Metrazol" in the treatment of the "functional" psychoses, Bernard Skorodin (*The Journal of Nervous and Mental Disease*, February, 1942) found that "Coramine", if given in sufficient quantity, would produce an artificial convulsion. He has recorded his treatment of twenty-one patients by this method. From 10 to 25 cubic centimetres of "Coramine" solution were used to provoke the fit. Four patients achieved remissions. There were no complications in the treatment; but the author, while believing that "Coramine" has certain advantages over other convulsant drugs, does not suggest it as a substitute for "Metrazol". Smaller doses of "Coramine" produce "shock" without a convulsion, and this effect the author considers advantageous in certain cases. This shock may last for ten minutes or longer and may produce intense anxiety. Sleep may subsequently be produced by the intravenous administration of a narcotic.

Trigeminal Neuralgia.

TRIGEMINAL NEURALGIA has been treated by vaso-dilator drugs by W. E. Adams and William Robinson (*The Lancet*, November 8, 1941) on the assumption that it is due to an ischæmia. Seven patients were treated, and nicotinic acid was used in doses of from 50 to 75 milligrammes given four times daily. Case histories are given in detail. Improvement was noted in every instance; at times it amounted to complete relief. In several cases amyl nitrite was inhaled at the height of the paroxysm and relief was obtained within a few minutes. To prove the theory of the ischæmic origin of trigeminal neuralgia, vaso-constrictor drugs were administered with the result that benzedrine was found to provoke attacks and to increase the number and severity of them. In one case benzedrine failed to provoke an attack in a patient previously treated by nicotinic acid.

Vitamin B and E Therapy in Tabes Dorsalis.

SIMON STONE (*The Journal of Nervous and Mental Disease*, February, 1942), being dissatisfied with the results of routine antispecific and fever treatment of *tabes dorsalis*, reports upon eighteen cases in which the patients were treated by vitamins *B* and *E* for periods ranging from three months to two years. Seventeen of these patients also received intraspinal injections of from 10 to 50 milligrammes of thiamin chloride. The writer believes that *tabes dorsalis* results from the combination of spirochætal invasion and vitamin deficiency. He suggests that certain factors in vitamin *E* are of importance to the maintenance of normal myelinization of the central nervous system. Thiamin chloride is believed to be a safer substitute for arsphenamized serum and easier to administer. Its use with that of the vitamins mentioned may be concurrent with the usual fever therapy and arsenical medication.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on October 23, 1941, at Saint Vincent's Hospital, Sydney. The meeting took the form of a number of clinical demonstrations by members of the honorary medical staff of the hospital. Part of this report appeared in the issue of May 9, 1942.

Bilateral Mammary Hyperplasia.

DR. C. F. A. DE MONCHAUX showed an unmarried woman, aged twenty-four years, who first presented herself at the radiotherapy department in November, 1940. She then complained of "lumpy swellings" in both breasts, together with mammary pain, both before and during her menstrual periods, of about twelve months' duration. The patient stated that she had had five minor operations on both breasts. These were possibly mastotomies for the removal of cysts.

On examination the nipples of both breasts were found to be inverted, and both breasts were somewhat tense and showed a number of typical areas of mammary hyperplasia (cystic variety), the left breast being thicker and more hyperplastic than the right. No clinical evidence of malignant disease was detected, and no axillary lymph nodes were palpable. A moderate course of X-ray therapy was given to both breasts (slightly more to the left) from December 3 to December 19, 1940. The patient was seen again early in February, 1941, when some improvement was found in the condition of both breasts. There had been no premenstrual pain since the X radiation, but she complained of scanty menstruation.

Two months later (April, 1941) the patient presented herself again for observation, complaining of some recurrence of mammary pain prior to two menstrual periods. The condition of the breasts was, however, much better, being then almost normal. It was then decided to give some more X-ray therapy, to be combined with hormonal therapy, in the form of stilbœstrol (œstrogenic hormone); this form of mastopathy (mammary hyperplasia) was now regarded as being hormonal in origin, due to ovarian dysfunction—a relative deficiency of the œstrogenic component. A second course of X radiation was given to both breasts, from May 1 to May 28, 1941, and stilbœstrol was administered orally *pari passu* with radiotherapy, without any gastric disturbance.

When seen again in June, 1941, the patient felt much better and had had no pain for some weeks. Improvement in the mammary condition was obvious; a slightly cystic condition of the right breast was still present. At her last visit to the department, in August, 1941, the patient felt well and had had no pain since the previous menstrual period. At the time of the meeting the patient felt quite well, but still had slight pain in the breasts during her menses. Apart from two or three small areas of residual hyperplasia, the condition of both breasts was normal. The breasts were no longer tense and the nipples were now everted.

Dr. de Monchaux commented that these cases of simple mammary hyperplasia occurred mostly in young unmarried women in the third and fourth decades of life, and that the primary treatment in all such cases of mammary hyperplasia was now X-ray therapy, accompanied sometimes by hormonal therapy (œstrogenic factor). The hyperplastic portion of the breast tissue was relatively radio-sensitive (as in the case of practically all hyperplastic states occurring in body tissues), and approximately 80% to 90% of all patients with mammary hyperplasia responded well to X-ray treatment. The patient shown had been successfully treated by the combined method of X-ray and hormonal therapy; both were logical methods of treatment in this condition, the hormone attacking the cause, and the X rays the effect of the disease. Dr. de Monchaux added that in many cases of mammary hyperplasia it was advisable, especially when the breasts were heavy and pendulous, for the patient to wear some form of comfortable support. Dr. de Monchaux concluded that there appeared now to be no indication whatsoever for operative interference, in the form either of a mastotomy or a mastectomy, in cases of simple mammary hyperplasia, the treatment by radiotherapy being as a rule satisfactory.

Contractures following Burns.

DR. V. J. KINSELLA showed a child, aged seven years, who had contractures following burns. The problems of treatment and prevention were discussed.

Epithelioma of the Pinna.

Dr. Kinsella next showed a male patient, aged eighty-five years, who had an advanced epithelioma of the pinna, with enlarged fixed glands in the neck and in the parotid region. Under local anæsthesia radical surgical removal had been carried out eighteen months previously, followed by deep X-ray therapy. A small recurrence was removed under local anæsthesia six months prior to the meeting. A large recurrence had appeared and developed during the last two months. Dr. Kinsella said that the case illustrated the palliative use of radical surgery even in advanced age. The patient had been comfortable and well until the last recurrence. Another interesting feature of this patient was a widespread melanoma of the face, which had been present for thirty years.

Carcinoma of the Colon.

Dr. Kinsella also showed a female patient, aged sixty years, who had had an advanced carcinoma of the colon involving the whole of the ascending and part of the transverse portions. It had been easily visible, even when the patient was dressed. It was adherent to the anterior abdominal wall. The mass had been removed nineteen months earlier, with the terminal coil of ileum, the ascending colon and half the transverse colon, and the deep layers of the anterior abdominal wall. The patient had regained her weight and her physical strength, and there was no sign of recurrence of the growth. Dr. Kinsella said that the case illustrated the fact that the largest growths were often less malignant than the smaller types.

Pathological Specimen.

Dr. Kinsella finally showed an appendico-sigmoid fistula with abnormal retroperitoneal fixation of the sigmoid loop in the right iliac fossa, and the ileo-colic and neighbouring branches of the superior mesenteric artery lying in front of the loop. The patient had undergone exclusion gastrectomy for a large duodenal ulcer which was penetrating the liver and pancreas. The appendix was then found to be fixed and thickened and it was removed; the removal began from the base, because the tip was fixed. The removal of the appendix led to a retroperitoneal cavity, lined by mucosa, identified later at autopsy as the sigmoid colon. A tube was tied into this. Adhesions formed around the track and the fistula was successfully shut off from the peritoneal cavity. The patient died three months after his operation from a persistent low-grade pneumonia.

A MEETING of the New South Wales Branch of the British Medical Association was held on April 23, 1942, at the Royal Alexandra Hospital for Children. The meeting took the form of a number of clinical demonstrations by members of the honorary medical staff of the hospital.

Partial Hemihypertrophy with Cœliac Disease.

DR. D. G. R. VICKERY showed a female patient, aged two years, who had been admitted to hospital on November 12, 1941; the right side of her body had from birth been larger than the left side; in particular the right leg was much larger than the left leg. Three weeks before her admission to hospital pronounced abdominal distension, ascites and œdema of the trunk and limbs developed.

On examination it was found that the child's right leg was grossly œdematous and elephantine, and the right arm was also œdematous, but less so than the leg. Slight œdema of the left arm and leg were noted. The abdomen was large and protuberant and ascites was pronounced; the wall of the trunk was œdematous. The child was pale and her skin was harsh and dry. The urine contained an amount of albumin varying from a faint trace to a large quantity on five days during her first three weeks in hospital under observation. Since then albuminuria had been detected on rare occasions. The urine contained no casts and no erythrocytes, and it was sterile. A blood count gave normal results, and no parasites were found in the stools. The blood urea content was 27 milligrammes per 100 cubic centimetres and the blood cholesterol content was 100 milligrammes per 100 cubic centimetres. The total protein content of the blood was 5·02 milligrammes *per centum*, 2·95 milligrammes being albumin and 2·07 milligrammes globulin. In view of these findings, the œdema was considered to be of nutritional rather than nephritic origin.

By December 4 the œdema had disappeared from the limbs and trunk, and the child exhibited the typical picture of partial hemihypertrophy. At this stage the stools were noticed to be large, bulky, pale and of the consistency of porridge. The total fat content amounted to 48% of the dried fæces; 98·1% of the total fat was split and 1·9% was unsplit.

Dr. Vickery said that the child had been given a diet poor in fat and rich in protein, with thyreoid extract, hydrochloric acid and vitamin *B*. Her general condition had greatly improved, and all œdema had disappeared from the normal left side. The abdomen remained distended, the buttocks were wasted and the stools still tended to be of cœliac type.

Congenital General Paralysis of the Insane.

Dr. Vickery also showed a boy, aged eleven years, who at the age of eight years had been examined in the out-patient department because from the age of five years he had not done well at school. He was "nervy" and babyish. He was an only child, born at term. He had walked at the age of twenty months and talked early. He seemed to develop to the age of five years, and then the mother noticed that he failed to concentrate and was mentally unstable.

On examination the child was well grown for his age. Frontal and parietal bossing was present. The nasal septum was sunken, the jaws were deformed and there was a tendency to peg teeth. The pupils were unequal, but reacted to light and accommodation. The knee jerks were much exaggerated, the ankle jerks were somewhat exaggerated, and Babinski's phenomenon appeared to be elicited. The child walked well and no ataxia was present. The external genitals were small. His mental state was most unstable; he cried one minute and laughed the next. He was talkative, talking intelligently and otherwise.

The cerebro-spinal fluid was examined. It contained 14 lymphocytes per cubic centimetre, and it was sterile. The fluid contained glucose and globulin; the chloride content was 760 milligrammes and the protein content 100 milligrammes per 100 cubic centimetres. The fluid reacted to the Wassermann test. The blood serum of the mother, father and child reacted strongly to the Wassermann test.

Dr. Vickery commented that the child had been given antisyphilitic treatment with "Bismol Neokharsivan" for two years, but his condition had not improved; steady mental deterioration was taking place.

Ectopia Vesicæ.

DR. H. G. HUMPHRIES showed two patients with *ectopia vesicæ*; one was a girl, aged seven years, and the other a boy, aged two years. The boy had been admitted to hospital for surgical treatment. After preliminary investigation it was decided to transplant the ureters into the sigmoid colon in separate stages. On August 7, 1941, the right ureter was transplanted into the sigmoid colon just above its junction with the rectum. On September 19, 1941, the left ureter was transplanted into the sigmoid colon some distance above the insertion of the right ureter. The patient was discharged from hospital on October 14, 1941, in good condition, being able to retain the urine in the bowel for periods of one hour or more. Later, on February 16, 1942, he was readmitted to hospital for removal of the bladder and plastic repair of the accompanying epispadias according to the method described by Ladd and Gross. He was finally discharged from hospital on April 10, 1942. The recorded concentration of urea in the blood on February 25, 1942, was 40 milligrammes per 100 cubic centimetres.

Hirschsprung's Disease.

Dr. Humphries also showed a female patient, aged five years, suffering from Hirschsprung's disease. On April 8, 1942, a left lumbar ramisection was performed, consisting of division of the medial branches from the left lumbar sympathetic chain with section of the sympathetic trunk below the lowest ganglion. Since operation the bowels had already acted normally on two occasions, for the first time in the patient's life. Within a few days a similar operative procedure would be adopted on the right side.

Dr. Humphries's third patient was a male, aged three months, suffering from Hirschsprung's disease. On March 26, 1942, left lumbar ramisection was performed, and on April 17, 1942, right lumbar ramisection.

Dr. Humphries remarked that in such cases of congenital megacolon improvement was to be expected by perseverance in post-operative bowel education over a period of many months.

DR. P. L. HIPSLEY showed a female patient, aged four years, who had been admitted to hospital on March 18, 1942. The mother said that the child had never been well since the age of one month; the abdomen had always been large and the bowels constipated. She had a small motion every day and the abdomen was growing larger.

On examination the child was somewhat thin and pale. The abdomen was much distended, and peristalsis was visible. X-ray examination after a barium meal revealed Hirschsprung's disease involving that part of the colon proximal to the sigmoid. At operation on March 24 left lumbar ramisection was carried out. The first, second and third lumbar ganglia were isolated and the medium division was removed with the ganglia and chain.

Contractures following Scalding.

DR. H. G. HUMPHRIES then showed a male patient, aged two years. He had been admitted to hospital with a history of having been scalded on both hands when nine months old; severe contractions of the ring and little fingers of the right hand had resulted. On October 30, 1941, a tubed pedicle graft was fashioned on the abdominal wall. On November 13, 1941, the proximal end of the graft was implanted on the palm of the right hand, the arm being fixed in position by a small plaster jacket. On March 13, 1942, the tubed graft was separated from the abdomen, opened and split to be adapted to the palm, ring and little fingers as required after removal of the scar tissue from the affected area. The muscle tendons were now able to glide comfortably under the thick graft, which had replaced the dense scar tissue.

Developmental Defect.

Dr. Humphries finally showed a female patient, aged one year, who had been admitted to hospital on account of a "congenital pectoral defect", described by John Thomson as occupying an area of the chest wall which might be covered *in utero* by the child's fist or forearm when the elbow was flexed. In the case under discussion a dwarfing of the hand and fingers and webbing of the latter were present, in addition to partial absence of the greater and lesser pectoral muscles, and also of the corresponding mamma and anterior ends of the fourth and fifth ribs. Plastic operations had been performed on the webbed fingers.

Adhesions following Intussusception.

DR. P. L. HIPSLEY showed a male patient, aged four years, who had first been admitted to hospital on November 18, 1940, when aged eighteen months, with an intussusception. Operation was performed, and a recently reduced colico-colic intussusception was found. Recovery was uneventful.

The child was readmitted to hospital on April 12, 1942; he was reported to have cried with abdominal pain for three days. No vomiting or fever was present, and the bowels had been opened the day before. A barium meal examination revealed no abnormality.

Cystic Hygroma and Lymphangioma.

Dr. Hipsley also showed a male patient, aged one year, who had been admitted to hospital on February 23, 1942. He had had a swelling on the right side of his neck since birth; the swelling was not increasing in size.

On examination a large, soft, cystic swelling of the right side of the neck and extending into the right cheek was found; in the cheek some dilated capillaries were present. At operation on February 24 portion of a cystic hygroma was dissected out, and deep X-ray therapy was given to the lymphangioma of the face. Pathological examination of the excised tissue verified the diagnosis of cystic hygroma.

(To be continued.)

Hospitals.

THE ROYAL PRINCE ALFRED HOSPITAL.

A MEETING of the Medical Board of the Royal Prince Alfred Hospital, Sydney, was held on February 12, 1942. The meeting took the form of a series of clinical demonstrations.

Hysterical Blepharospasm.

DR. J. A. MCGEORGE showed a female patient, aged sixty-five years, suffering from hysterical blepharospasm. For the past twelve months she had suffered from increasingly severe spasmodic contractions of the *orbicularis oculi*. She complained of a strange sensation coming from the back of her neck to affect her eyes. She said that her eyes ached and felt as if she had sand in them. The spasm was persistent and quite obvious. No history was obtainable

which threw light on the cause of the disability. Apart from some mild financial worry, she had been subjected to no form of mental stress. Her eyes were bandaged for one month, but this had had no effect on the condition.

Examination revealed a typical blepharospasm. The patient complained of tenderness on pressure over the spine of the second thoracic vertebra. The fundi, which as might have been expected were examined with considerable difficulty, were normal. No other physical signs were detected.

Dr. McGeorge said that such conditions represented psychologically an attempt to avoid some unpleasant situation. A typical case was that of a railway man who witnessed a nasty accident and subsequently developed blepharospasm—an obvious attempt to avoid a repetition of his unpleasant experiences. The treatment was psychological, and an endeavour was made to determine the factor that produced the condition originally and to reassure the patient. In the case under discussion little success had been met with. Dr. McGeorge hoped that no one would suggest hypnosis as a form of treatment; it was difficult enough to induce the patient to keep her eyes open.

Atypical Dementia Paralytica.

Dr. McGeorge also showed a female patient, aged forty-seven years, suffering from atypical *dementia paralytica*; she showed the mental symptoms of an ordinary cerebral degeneration without the characteristic fatuous, expansive, grandiose outlook of such patients. Her condition had originally been diagnosed as myxœdema, and she had been treated with thyreoid extract. She was unable to give a coherent account of herself. She was dull, apathetic and emotionally inert and unresponsive. Her speech was somewhat slurred. She exhibited Rombergism and her gait was unsteady. Her reflexes were exaggerated, her pupils reacted sluggishly to light, her tongue was tremulous. She showed no signs of thyreoid deficiency; her skin was moist, her hair was greasy and her slowness of mentation was due to her physical condition. The Wassermann and Kline tests produced positive reactions with the blood serum. The cerebro-spinal fluid reacted to the Wassermann test, and the result of the Lange gold test was "4555322100". The test for globulin produced a "triple positive" result and the total protein content was 100 milligrammes *per centum*.

The patient was inoculated with malaria and had 12 rigors; they occurred every day and sometimes twice a day. At the end of this treatment she was obviously extremely anæmic, and a saturated solution of iron was administered. She responded rapidly to this and became quite manic, being garrulous, foolish and irresponsible, exemplifying much more typically the condition from which she suffered. Dr. McGeorge said that it was doubtful at the time of the meeting whether it could be claimed that she had derived any benefit from the treatment she had received; to convert an atypical into a typical *dementia paralytica* could hardly be regarded as a noteworthy achievement.

Correspondence.

THE STORY OF MATTHEW FLINDERS.

SIR: Your reviewer has stated that "in no instance has the author played with history". I amused myself by reading the book critically. It is possible that others may be interested if I recount some of the historical inaccuracies that I found. Before doing so, I wish to make it plain that the book is in my opinion too a great one, and that the inaccuracies are quite excusable as a rule in the making of such a fine story. The gifted authoress has (in a phrase she used herself in the book) been able "to marry fact and fancy".

For the purposes of her story she has made Ann Chappell nine years younger than she was; actually she was four years older than Matthew Flinders. Uncle John was really only eight years his senior, though George Bass was eleven years older than Matthew. At the time when grandfather Flinders was making "his long white beard a landmark" he was really only fifty-two years of age after all. Other minor discrepancies in ages are present in the book, but they are immaterial.

A curious error appears on page 77. The reference to Watt's kettle is out of place, because the rotatory steam engine was set up in 1784 and there were many metals known other than silver and gold. Two pages later, where the authoress is describing the year 1792, I think she hints at the previous occurrence of the battle of St. Vincent, which actually did not take place until 1797. Another anachronism is on page 165, for Moby Dick was Herman Melville's creation in 1851. On the following page, Bass, in conversation with Flinders, is reputed to say that Wilson's Promontory "sheltered us from the venom of the strait"—but he had not then found it to be a strait. On page 172 the authoress has repeated the fiction that Maria Island was named after Tasman's sweetheart; it was really named for Van Diemen's wife; they did not have a daughter Maria.

Ernestine Hill has taken liberties with the correspondence known as the Flinders Papers and with other letters. She has used phrases from correspondence and represented them as conversations. An instance of this is the account of Ann's conversation with Betsy on her wedding day; Betsy was not even at Donington on that day. It is true that Ann wrote these words to Betsy on that day, but the authoress has left out the following phrase: "It grieves me much thou art so distant from me." Similarly, the circumstances surrounding Matthew's separation from Ann, when he left her behind him, have been exaggerated. The material has been derived from a series of letters between Banks and Flinders. St. Vincent and Spencer found her "seated in the Captain's cabin without her bonnet"; the kissing scene is an invention! Flinders did not say: "I shall give up the wife for the voyage of discovery"; he wrote to Banks: "Mrs. Flinders will return to her friends immediately that our sailing orders arrive." At the time of sailing Ann was ill at her father's home in Lincolnshire and was not there to say good-bye.

Thistle's toast to "Australia" is not historical. Flinders used the word for the first time in a letter written to his brother Samuel on August 25, 1804: "I call the whole island Australia or Terra Australis"—he did not know of its previous use—and he also used the name in "A Voyage to Terra Australis" in 1814 (see page 459).

On page 307 Ernestine Hill has omitted to refer to the fact that Flinders applied officially to Decaen to be allowed to go to the Maison Despeaux. On page 373 the inference is that Elder, Matthew's faithful servant and companion, did not leave him until 1807, but really he left in June, 1806.

Your readers may be interested to note that Napoleon's order for Flinders's release was signed in France on March 11, 1806, and was not received in Ile de France (Mauritius) till July 20, 1807, and his sword was restored to him on June 13, 1810. He was six years five months and twenty-seven days in captivity and arrived in England on October 23, 1810, after an absence of nine years three months.

Yours, etc.,

H. BOYD GRAHAM.

Melbourne,
May 8, 1942.

RICKETS IN AN AUSTRALIAN COMMUNITY.

SIR: I cannot refrain from congratulating Dr. Clements upon his careful observation, thorough review and sane deduction concerning the incidence and nature of rickets in Canberra. It was particularly interesting for me to note that he confirms the high percentage of this disorder which I reported as existing among infants of the poorer classes in Sydney ten years ago. Dr. Clements's figures, compiled after a more prolonged study and with better radiographic facilities, are more reliable than mine. Clinical evidences of rickets were apparently less obvious than among Sydney slum infants. I am sorry that Dr. Clements did not choose to publish his radiographs or more biochemical evidence, for example blood phosphatase values of the existence of rickets in his infant community, in order to convince the sceptics who still abound, even amongst the most erudite.[1] One reason for this mental dysphagia is the undoubted fact that the interpretation of mild rickets, and particularly the differentiation from physiological post-natal calcium redistribution in the metaphysis, requires a little special study uninfluenced by the pictures of florid rickets shown in the text-books. Dr. Clements states that the duration of rickets in his series was very short in a high percentage of instances, presumably because these children were promptly treated. In centres less alive to the prevalence of the defect, many children probably relapse during the first two winters of their existence and form a high proportion of the knock-kneed youngsters so common today, besides providing an assured future for those of their own generation who later follow the profession of dentistry.

The baby clinics of New South Wales have performed such an excellent service to the community that they have

[1] Dr. Clements submitted radiographs for publication, but he agreed that these should not be published owing to the need for conservation of paper.—EDITOR.

diverted many a would-be pædiatrician to more economically safe occupations. But they, in common with many doctors, appear to think that as long as a baby is breast fed, gains weight according to the standard scale, and shows an average resistance to infection, all is well. The possibility that rickets in any form can be found consistently among the breast-fed progeny of the well-to-do is discounted. We know now that avitaminosis, anæmia and mineral insufficiency can insidiously undermine the health of the offspring of both duchess and dairymaid. It is sincerely to be hoped that sisters in charge of these centres will be interested in the clinical, and even the radiographic recognition of mild rickets. X-ray facilities and hæmoglobinometers are not superfluous at any baby clinic, and the abandonment of cod liver oil emulsion for accurately measured doses of vitamin *D* and calcium in a cheap form as prophylactics is long overdue.

The nature of my late and present practice does not authorize me to enlarge further upon this important subject, but does not deny me the earnest hope that the lessons of Dr. Clements's careful work will not be overclouded by current problems of national medicine or impeded by any political resistance.

Yours, etc.,

J. KEMPSON MADDOX,
Surgeon Lieutenant-Commander,
Royal Australian Naval Reserve.

Undated.

The Royal Australasian College of Surgeons.

MEETING OF CENSORS.

THE next meeting of the Australian Board of Censors of the Royal Australasian College of Surgeons will be held at the college, Spring Street, Melbourne, probably in September, 1942. Candidates who desire to present themselves at this meeting should apply to the Censor-in-Chief for permission to do so on or before June 30, 1942. The appropriate forms are available at the college, Spring Street, Melbourne, and at the offices of the various State secretaries.

Obituary.

RICHARD SANDERS ROGERS.

WE are indebted to Professor J. Burton Cleland, Adelaide, for the following appreciation of the late Dr. Richard Sanders Rogers.

By the death at Adelaide on March 28 of Richard Sanders Rogers at the age of eighty, the medical profession has lost another of those outstanding individuals amongst its members who in their leisure time have added much to the knowledge of Australian natural history. He takes a worthy place beside such medical naturalists as Dr. George Bennett, Dr. James Cox and Dr. E. W. Ferguson of Sydney, Dr. W. D. K. MacGillivray of Broken Hill, Surgeon Lieutenant-Commander W. E. J. Paradice, and Sir Joseph Verco and Dr. R. H. Pulleine of Adelaide. Beginning with a general interest in botany, fostered by his association with Professor Ralph Tate, Dr. Rogers early paid particular attention to the orchids, first of all those of South Australia and then, as his grasp of a difficult branch of botany developed, of Australia as a whole, of the adjacent island of New Guinea and even of the Pacific Islands in the Australian sphere. He thus became the world authority on orchids for the Australasian region. With Mrs. Rogers, herself a keen collector and an ever-helpful assistant, Dr. Rogers visited from time to time all portions of South Australia where orchids grow. The arrival of a new find from an amateur collector, schoolmaster or local field naturalist at once sent the two off to search for further specimens of the growing plant. As a result of his botanical work, Dr. Rogers was elected a Fellow of the Linnean Society of London in 1924. In 1911 he published an "Introduction to the Study of South Australian Orchids". He contributed the portion, consisting of 40 pages, dealing with the Orchidaceæ in Part I of J. M. Black's "Flora of South Australia", published in 1922, and the article on "Orchids" for "The Australian Encyclopædia", 1926, edited by Jose and Carter. In Black's Flora, of the 98 species and three varieties of orchids therein recorded for South Australia, 17 species and two varieties had been originally described and most of them discovered by Dr. Rogers. Beginning in 1906 in Volume 30 and ending in 1940 with Volume 64, Dr. Rogers contributed 25 papers to the *Transactions of the Royal Society of South Australia* as well as two other papers in collaboration, all dealing with orchids. He contributed the following two papers to the *Proceedings of the Royal Society of Victoria* (New Series): "Notes on Certain Species of Pterostylis" (Volume 28 (1), pages 105-111, plates 7-9, 1915) and "Chiloglottis Pescottiana sp. n." (Volume 29 (2), pages 139-141, plate 25, 1918) and to the *Journal of the Royal Society of Western Australia* one entitled "A New Genus of Australian Orchid" (Volume XV, page 1, plates 10 and 11, 1928-9). Amongst the most interesting species described in these papers is *Rhizanthella gardneri*, a Western Australian orchid that actually flowers underground. Some years ago he submitted two reports to the Government of South Australia, one entitled "A Medical Survey of the Feeble-Minded in South Australia" and the other "A Preliminary Medical Survey of South Australian School Children". I cannot find any record of these having been published. Dr. Charles Fenner, Director of Education, South Australia, informs me that in 1910 the government printer, North Terrace, Adelaide, printed a "Report on the Medical Inspection of One Thousand Pupils attending the Public Schools of South Australia during 1909", by R. S. Rogers, M.A. (Adelaide), M.D. (Edinburgh). A copy has been lodged in the Public Library, Adelaide, by the Director of Education. Dr. Rogers also published in the *Education Gazette*, South Australia, "Growth and Development of the Child" (November, 1907, page 206) and "Maladies of Children—from the Teacher's Standpoint" (November, 1907, page 256).

Dr. R. S. Rogers was born in Adelaide in 1862. His father was Joseph Rogers. In 1887 he married Jean Scott Paterson, of Edinburgh. He graduated as Bachelor of Arts in the University of Adelaide in 1883, and at the time of his death was its senior graduate. He obtained the degree of Master of Arts in 1897, and in 1936, at the age of seventy-four, and fifty-three years after his first graduation, that of Doctor of Science in the same university awarded on the merit of his published researches on the orchids. After obtaining his B.A., Dr. Rogers went to Edinburgh to study medicine, being senior medallist of the year in zoology in 1884 and graduating as Bachelor of Medicine and Master of Surgery at the University of Edinburgh in 1887. He took his doctor's degree in medicine at Edinburgh in 1893 and at Adelaide in 1897. He was registered by the Medical Board at Adelaide on May 16, 1888. It has not been possible to ascertain when he was elected a member of the South Australian Branch of the British Medical Association, but his name appears, apparently for the first time, as a member present at a meeting on November 19, 1891. As he resigned membership at the end of 1938, he had been a member of the Branch for over fifty years. Dr. Rogers was appointed lecturer in forensic medicine at the University of Adelaide in 1919 and held this position till 1939. He was a member of the Board of Management of the Adelaide Hospital from 1896 to 1922, and during the years 1913-1921 he was deputy chairman. He was honorary physician at the Adelaide Hospital from August 20, 1897, to February 28, 1909, and honorary consulting physician from March 1, 1909, to the date of his death. He was a member of the Medical Board of South Australia from January, 1911, to May, 1940, and president from June, 1932, to December, 1937. At the Enfield Receiving House he was superintendent (visiting) from June 15, 1922, till the end of 1936, and deputy superintendent (visiting) from January 1, 1937, to December 31, 1938; at the Northfield Mental Hospital he was superintendent (visiting) from September 5, 1929, till the end of 1936; and he was honorary consulting psychiatrist to mental institutions from April 20, 1939, till the date of his death. He was a member of the Board of Governors of the Public Library, Museum and Art Gallery for forty-four years, and president from 1929 to 1931, president of the Royal Society of South Australia in 1921-1922, vice-president from 1914-1919 and from 1922-1924, and was a member of council from 1907-1914 and from 1919-1921, president of the Justices' Association, and for a short time acting city coroner. He served in the South African War and as lieutenant-colonel was in charge of Keswick Base Hospital from 1914 to 1919.

Dr. Rogers's quiet and gentle manner must have been very soothing to the mentally afflicted and borderline cases that came under his care at the Enfield Receiving House. He was a delightful and interesting companion on collecting expeditions in the bush, as the writer can bear testimony. His lectures on forensic medicine were unusually full of valuable information specially applicable to South Australia

and are still in use, the Medical Students' Society having had multiple copies typed for the benefit of its members. As lasting memorials in his honour, more enduring than brass, two Australian orchids have been named after him by their describers, the green-hood *Pterostylis Rogersii* Coleman and *Prasophyllum Rogersii* Rupp.

Australian Medical Board Proceedings.

SOUTH AUSTRALIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1919 to 1935, as duly qualified medical practitioners:

McPhie, John Milroy, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Grant, Donald Kerr, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Hunter, Geoffrey Allan, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Verco, Peter Willis, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Jeffries, John Singleton, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Tippelt, Ferdinand Marcel, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Gurner, Colin Marshall, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Zimmet, Jacob, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Guthaner, Ernst, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Sheppard, Mark Yeatman, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Cheek, Nancy Olive, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Gordon, John Vivian, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Yates, Percy Cocker, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Davies, Ellice Jean, M.B., B.S., 1915 (Univ. Melbourne), Ernabella, via Oodnadatta.
Kerr, Challen Sydney, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
McGlashan, John Gardner, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Chambers, William Charles Teesdale, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Heddle, Robert Charles, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Dinning, Trevor Alfred Ridley, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Texler, Karl Maria, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Martin, Ian Holland, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Hammill, Robert Duncan, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Böer, Edward Douglas, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Rosenberg, Mendel Wolf, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Clarke, Miles de Courcy, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.
Rowe, Allan Gordon, M.B., B.S., 1942 (Univ. Adelaide), Adelaide.

Medical Appointments.

The undermentioned have been appointed Resident Medical Officers at the Royal Adelaide Hospital, Adelaide, South Australia: Edward Douglas Böer, William Charles Teesdale Chambers, Nancy Olive Cheek, Miles de Courcy Clarke, Trevor Alfred Ridley Dinning, John Vivian Gordon, Donald Kerr Grant, Colin Marshall Gurner, Robert Duncan Hammill, Robert Charles Heddle, Geoffrey Allan Hunter, John Singleton Jeffries, Challen Sydney Kerr, Ian Holland Martin, John Gardner McGlashan, John Milroy McPhie, Allan Gordon Rowe, Mark Yeatman Sheppard, Peter Willis Verco, Percy Cocker Yates, Barbara Quinn Young.

Dr. Gerald Darnton Talbot Watson, Dr. Alan Boswell Hewitt, Dr. William George Farrell and Dr. Albert Antliff Weir have been appointed Public Vaccinators at Camperdown, Cranbourne, Pakenham and Terang, Victoria, respectively.

Dr. Robert Jeffery Long has been appointed a Public Vaccinator in the Department of Public Health, Victoria.

Dr. John Catarinich has been appointed Director of Mental Hygiene of Victoria for a period of five years from May 1, 1942, pursuant to the provisions of the Lunacy Acts.

Dr. G. B. R. Wooster has been appointed a Member of the Board of Directors of the Orange Base Hospital, New South Wales, for a period of three years from April 15, 1942, according to the provisions of the *Public Hospitals Act*, 1929-1940.

Diary for the Month.

MAY 20.—Western Australian Branch, B.M.A.: Branch.
MAY 22.—Queensland Branch, B.M.A.: Council.
MAY 28.—South Australian Branch, B.M.A.: Branch.
MAY 28.—New South Wales Branch, B.M.A.: Branch.
MAY 29.—Tasmanian Branch, B.M.A.: Council.
JUNE 3.—Western Australian Branch, B.M.A.: Council.
JUNE 4.—South Australian Branch, B.M.A.: Council.
JUNE 5.—Queensland Branch, B.M.A.: Branch—Joseph Bancroft Memorial Lecture.
JUNE 9.—Tasmanian Branch, B.M.A.; Branch.
JUNE 12.—Queensland Branch, B.M.A.: Council.
JUNE 17.—Western Australian Branch, B.M.A.: Branch.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility unless such a notification is received within one month.

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